

OBSTETRIC VIEWPOINT ON CEREBRAL PALSY

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Modern organisation and techniques of obstetrics have largely eliminated the dangers to the baby of mechanical birth trauma and of late pregnancy anoxia. There is still, however, a great deal that the obstetrician and gynaecologist can contribute in preventing damage to the baby. He should first of all accept that cerebral palsy, epilepsy, mental defect, etc. are problems within his direct field. Because of his interest in infertility, abortion, pregnancy and labour, he should be a leader in research studies directed towards the investigation of the aetiology of such defects, and, therefore, assist in formulating techniques to minimise such dangers.

Most of the current inadequate information is due to poor clinical records and to lack of appreciation that there are aberrations in pregnancy and labour, of little apparent importance at the time, which can affect the subsequent development of a child.

Introduction

Within this century the world population will be doubled, and many more people will live into old age. This has become possible through the application of advances in public health and paediatrics, and, to a less extent, in general medical care. There is no shortage of human beings. Despite this, modern medical science is acutely concerned with wastage in reproduction, whether that wastage arises from infertility, abortion, perinatal loss or the birth of a handicapped child.

The community is already facing great social problems from the host of young people born in the war years. These problems will increase numerically when the "bulge" children (of 1946-48) reach adolescence. In addition to the relatively simple problems of housing, employment and education, we have the much graver problems set by the mental or physical defective and by the delinquent, who are bound to appear in at least equal incidence but in greater numbers than in the past.

To the individual parents, the tragedy; to the community, the diversion of much time, money and effort in the care and supervision of the handicapped.

There is, however, another side to this problem. We are desperately short of able people. Only some 10% of our children reach university entrance levels. We cannot afford damaged children, even if the damage is so slight as to make them drop below the top 10% level of attainment. From the professional, humanitarian, social and community viewpoint, therefore, pregnancy wastage, in its broad sense, must be studied and its causes elucidated.

The Obstetrician's Viewpoint

Human reproduction should be the field in which the obstetrician/gynaecologist is pre-eminent. This rather obvious statement needs emphasis at this time when so much of the really valuable work is contributed from other disciplines.

I would agree that the geneticist, social scientist, cell chemist, and embryologist have knowledge and techniques to ex-

plore their specific areas: and that the paediatrician and child psychiatrist are skilled in the care and investigation of the child. However, the obstetrician/gynaecologist, in his interest in infertility, abortion, pregnancy and labour, must serve to correlate and apply the new knowledge. He has to treat, advise and assess; to act, or hold his hand, according to the sum of his information.

He must not, however, think of himself only as a well-informed clinician applying the knowledge of others, or even as a correlator of available knowledge. He must himself study, at a research level, some aspects of the problem, and try to become a leader, or at least a member, of a research team.

The obstetrician's interest, therefore, in pregnancy wastage, covers all known anomalies of human reproduction. I would like, however, in this paper, to consider, first, those major factors in pregnancy and labour which are of known importance in the aetiology of "cerebral palsy". This approach must, however, be speculative without the detailed factual information which can be obtained only from extensive prospective studies. Later, I would like to take a somewhat broad view of the research problem as I see it.

Birth Trauma.—This term must be confined to physical damage arising from mechanical difficulty in labour or delivery. In this sense, it is seen mainly in the babies of primigravidae over 25 years of age who are short in stature (under 5 ft. 4 in. in height). If the pregnancy is prolonged after term, or if there is malposition of the foetal head or a breech presentation, then the risk is greater still. In past years, the combination of prolonged pregnancy, prolonged labour and disproportion often led to anoxia of the foetal brain in addition to mechanical trauma. Birth trauma was preceded, and probably predisposed to, by an intracranial congestive stagnant

anoxia syndrome classically described by Holland (1922). Interference with venous return, vascular engorgement, oedema, and capillary haemorrhage produced local tissue damage and made vessels and septae more liable to tearing. Difficult mid-cavity forceps delivery, under indifferent anaesthesia, completed the risk.

Over the last few years, advances in obstetric practice have virtually eliminated mechanical birth trauma and the mechanical intracranial anoxia syndrome. Improvement in the health, nutrition and stature of women now in their child-bearing years, and a trend to earlier child-bearing, have improved the mother's state. The obstetrician now terminates difficult labour much sooner, either by Caesarean section or by forceps delivery, before serious moulding has occurred. He thus delivers the baby before it has become damaged (mechanically or by anoxia) and the methods of delivery themselves are much less traumatic. Anaesthesia has also improved, especially for the mother, permitting the wider application of surgical methods of delivery.

Anoxia.—The problem of anoxia in late pregnancy and during labour has been extensively studied in recent years, though a great deal remains to be settled. Studies by James *et al.* (1958) and Weisbrot *et al.* (1958) have confirmed the beliefs of Minkowski *et al.* (1953) and Walker (1955/56) that very many babies have a deficient oxygen supply at birth. In most this is a transitory state, but in others the anoxia is of longer antepartum or intrapartum duration, or persists after birth, and may lead to permanent damage.

A deficient supply of oxygen to the baby in late pregnancy and labour can arise from several and often multiple causes which are best considered under two headings:

- (a) Inadequate transplacental transfer.
- (b) Adequate placental transfer but interference within the child itself.

The first group, characterised by poor oxygen levels in the umbilical vein and artery, is seen, for example, in cyanosis-producing conditions in the mother, in accidental haemorrhage, severe pre-eclampsia, diabetes, renal disease, in many cases of prolonged pregnancy, occasionally after previous threatened abortion, and probably also after prolonged labour with frequent high-tone contractions.

The second group is characterised by adequate oxygen levels in the umbilical vein but low levels in the umbilical artery. James *et al.* (1958) have emphasised the findings of Dawes *et al.* (1954) and Dawes (1957) that the umbilical artery oxygen level, especially in times of stress, is representative of the oxygen levels in the blood reaching the foetal brain and heart. Van Slyke (1959) has recently stressed the importance of umbilical artery readings.

This situation is seen with cord strangulation or pressure, and in association with certain cases of foetal heart slowing (often with undue pressure on the head). It is classically seen when the head is held up on the perineum before delivery, or when the child is allowed to wait too long for delivery of the shoulders.

As with birth trauma, modern obstetricians interfere sooner in the presence of anoxia-producing conditions and certainly in the presence of foetal distress. Few would happily allow long periods of labour in the presence of meconium staining or with a slow or irregular foetal heart.

Prematurity.—In a recent survey in Eastern Scotland, Mitchell (1959) found that 27.5% of cerebral palsied children had been premature at birth (excluding cases where the damage was of postnatal origin). The prematurity-rate in this region of Scotland is at least 6.8% of all births. Cerebral palsy was therefore three to four times more likely after premature than after mature birth. This broad statement is, however, of no help in determining aetiology.

The premature baby, during pregnancy and labour, has been exposed to special risks:

- (1) Impairment of nutrition, oxygen supply, etc. owing to complicating maternal disease, local or general.
- (2) Threatened abortion earlier, and bleeding before or during labour.
- (3) Breech delivery, with the possibility of cord strangulation or prolapse.

Thus anoxia to a large extent, and trauma to a slight extent are special risks.

Post-prematurity cerebral palsy is of a distinctive pattern, however, and Polani (1958) has pointed out that it can rarely be explained on the same basis of trauma or anoxia in mature babies. Polani (1958) lists two extra "risks" of the premature:

- (4) That the maternal lesion, genetic or disease, responsible for the prematurity might also be responsible for cerebral defect or damage.
- (5) The postnatal risks peculiar to the premature.

It is tempting to assume that prematurity and defective cerebral development are linked, but it is specifically this type of supposition that is incapable of proof without a great deal more study.

The peculiar postnatal risks of the premature are, of course, very real. Born too soon, the premature baby has two problems:

- (1) His tissues must continue to mature and differentiate in a totally unsuitable environment of oxygen tension, blood-flow and basic nutrition.
- (2) His brain, lungs, heart, liver, gastrointestinal and renal tracts are called on for functions for which they are not yet ready. His red cells are not designed to off-load oxygen to his tissues at the new level of extrauterine demand.

Resuscitation.—Is this a problem for obstetrician, paediatrician, anaesthetist, or midwife? I am quite certain that all per-

sons concerned in "delivery" should appreciate the importance of resuscitation and have some knowledge of the necessary skills. Good obstetrics will produce fewer babies who need expert care. However, a baby's life and certainly his future well-being may well depend on the skill with which he is cared for in the first postnatal hour.

A paediatrician fully trained in the modern techniques of resuscitation should be available at all difficult deliveries, for all premature deliveries and for the unexpected difficult baby. Just as the obstetric registrar "lives in", so should the paediatric registrar.

Obstetrical Factors in the Aetiology of Handicapped Children

It is generally assumed that the factors I have discussed are important in the aetiology of cerebral palsy. This is certainly true, but we know nothing about how the damage is caused or how often it follows individual procedures. There is a great deal of room for extension of the many excellent follow-up studies of damaged or premature children into a full-scale study of mothers (and fathers) during pregnancy and labour, with follow-up of all children. It is, after all, the obstetrician who has to learn how to prevent damage and he must learn the obstetrical situations under which the damage occurs. The clinical obstetrical documentation of series of cases reported in the past is dismally inadequate. There are, as I see it, several reasons for the situation in which we now find ourselves:

- (a) Retrospective studies of pregnancy and labour are almost bound to fail because of inaccurate or incomplete records. How often would threatened abortion be mentioned in the notes? How often is the foetal heart carefully recorded throughout labour and up to the moment of delivery? Is meconium staining of the liquor always noted? Does the obstetrician or midwife record that the head was held up on the perineum for five minutes, that the shoulders were not born for several minutes (while the "next pain" was awaited), or that the mother was cyanosed with her gas/air machine? Would not accidental haemorrhage in late labour be written down as "excessive show", if it seemed worth while recording at all? As a rule, these and many other potent associations with foetal anoxia are not considered abnormal or important enough to record, except in a few hospitals where standards are very high.
- (b) In field studies, such conditions as cerebral palsy, epilepsy, behaviour defect and mental deficiency are lumped together, even though the aetiological causes vary widely from genetic anomalies, through inadequate nutrition of the mother in childhood, to immediate post-delivery anoxia. Mental deficiency is probably highly correlated with heredity and only rarely with pregnancy and labour, whereas cerebral palsy is probably less often genetically determined.
- (c) Cerebral palsy, epilepsy and mental defect are not single conditions. The recent definition of the "Little Club" (Polani 1959) may tighten the clinical diagnosis of cerebral palsy, but what of "epilepsy"? If we try to study the incidence of epilepsy in a follow-up study, do we include children with aberrant E.E.G. patterns, but no other clinical features, in the normal or abnormal groups? If we do not have E.E.G. studies, how can we assess incidence—that is, if we agree on the meaning and significance of aberrant E.E.G. patterns?
- (d) Pathological findings on the brains of handicapped children are rarely available and the clinical features of each case are rarely correlated with definite pathological lesions. It might be possible to define aetiology if full information were available on the clinical history, clinical neurological findings and detailed pathology. But neuropathology is as yet not sufficiently precise, and new methods of determining damage must be developed. A mental defective or palsied child must have damage or aberration somewhere and we must learn how to detect it.
- (e) One of the most complicating features is the possibility of multiple aetiological factors in the same case. Certain community groups are more prone to late pregnancy bleeding and to prematurity (the continuum of reproductive causality of Lilienfield and Pasamanick 1955). It is also those groups who are more likely to have a mechanically difficult labour. Moreover, it is exactly those groups who have a poorer genetic background and a higher incidence of mental deficiency and whose children, once born, have poorer social, physical and educational opportunities.

What can the Specialist or General Practitioner Obstetrician do now?

(a) He can recognise that "normal reproduction" is that standard achieved by some 20% of his patients and that all others fall below that level to a greater or lesser degree.

(b) He can improve his records of individual patients so as to include accurate information on history, pregnancy, labour and delivery. This is a very difficult task, especially when it is still rare for the same individual or team to care for a woman from very early pregnancy, through labour, until the end of the lying-in period.

(c) He can realise that a woman has a reproductive history either behind her or before her, and appreciate that behaviour in a given pregnancy and labour can often be foretold from the past obstetric history.

(d) He can improve the standards of care in pregnancy and labour, so that he can foresee difficulties and prevent them becoming serious. A patient, well cared for and observed antenatally, rarely becomes a real obstetric emergency.

(e) He can welcome the trend to specialist antenatal care and hospitalisation for delivery, which allows the wider application of his special abilities, but he must also try to avoid the risks associated with over-crowding of clinics and overwork of midwife and medical staff.

(f) He can realise that abortion and prematurity are really his problems. He should use his influence to ensure that adequate antenatal beds are provided to allow long-term rest to selected cases—e.g., from 28 to 34 weeks in twin pregnancy (especially in primigravidae and mal-nourished multigravidae), in cases of late pregnancy bleeding, etc., in an attempt to

prevent unduly premature delivery.

(g) He can ensure that the best available therapy is applied in labour and at the birth of a child in the risk situations—e.g., a child delivered before the 37th week or known to be small should be delivered by senior personnel with a paediatric registrar in attendance.

(h) He can welcome into his clinics, wards and labour suites experts in other disciplines who wish to study specific problems in the human, and can guide those studies from his specialist knowledge of the problem.

(i) He can himself take an active personal interest in some research aspect of the problems of reproduction.

The Future

Perhaps our standards are not high enough. Less than 10% of babies are lost in the perinatal period or are recognisably damaged or defective. Only some 10% reach university entrance levels of educational attainment. If those two groups are really the top and bottom 10% of our children, really the opposite ends of a "scatter", then what of the 80% in between? We know that anoxia is not an "all or none" insult and there must be degrees of damage. Does this hold for other causative factors? How many children are damaged, just a little, so that they are well below their potential level but not obviously defective? How important is nutritional and educational opportunity after birth in subsequent brain growth and development? Is it possible to measure basic and acquired intelligence or basic or acquired ability?

Those and countless other questions all to be answered by imaginative and all-out studies in the years to come.

SUMMARY

There is still, despite over-population, urgent need for elucidation of the aetiological factors of pregnancy wastage. The obstetrician/gynaecologist should be a leader in such studies.

Cerebral palsy is said to arise from mechanical birth trauma or from late pregnancy or intrapartum anoxia. Modern obstetrical techniques should lead to the disappearance of such cases. Prematurity is a much more difficult problem. The obstetrician must seek the causes and attempt to minimize the risks to the foetus.

Present research is handicapped by lack of available obstetrical information. Many and varied aetiological factors can cause the same lesion. In field research lesions of widely different aetiological background are studied together as if they were one syndrome.

Much research needs to be done. The obstetrician should lead, should welcome into his clinics workers from other disciplines, and should guide their studies in so far as clinical experience is essential.

RESUME

L'infirmité motrice cérébrale du point de vue obstétrical

Il y a encore, malgré une sur-population, un besoin urgent de mettre en lumière les facteurs étiologiques du gaspillage des grossesses. L'obstétricien/gynécologue est appelé à conduire une telle recherche.

On considère comme causes de l'infirmité motrice cérébrale les lésions obstétricales mécaniques, les grossesses tardives, l'anoxie pendant le travail. Mais il y a tout lieu de penser que l'obstétrique moderne aura raison de ces cas. La prématurité est un problème bien plus difficile. L'obstétricien doit en chercher les causes et essayer de réduire les risques encourus par le foetus.

L'absence de renseignements obstétricaux gêne considérablement la recherche actuelle. Une même lésion peut avoir été causée par des facteurs étiologiques nombreux et variés. Dans le secteur de la recherche, des lésions d'origine tout à fait différentes font partie de la même étude comme si elles ne formaient qu'un seul syndrome.

Un grand travail de recherches doit être fait. L'obstétricien doit guider et accueillir dans sa clinique les chercheurs de disciplines différentes et doit orienter leurs études dans le mesure où l'expérience clinique est le fait essentiel.

ZUSAMMENFASSUNG

Die zerebrale Kinderlähmung vom Gesichtspunkt der Geburtshilfe aus angesehen.

Trotz der Übervölkerung, bleibt es noch dringend nötig, die ätiologischen Faktoren der Vergeudung der Schwangerschaften zu erschliessen. Dem Geburtshelfer und Frauenarzte steht es bei, diese Forschungsarbeit zu leiten. Man nimmt als Ursachen der zerebralen Kinderlähmung mechanische Schädigungen, späte Schwangerschaften, Anoxämie während der Entbindung an. Man kann aber denken, dass die moderne Geburtshilfe solche Fälle überwinden wird. Die Frühgeburt ist ein viel schwierigeres Problem. Der Geburtshelfer muss ihre Ursachen suchen und versuchen, die Gefahren die der Fötus läuft, zu vermindern.

Die Abwesenheit obstetrischer Angaben

stört die jetzigen Forschungen beträchtlich. Dieselbe Schädigung kann durch viele und mannigfaltige ätiologische Faktoren hervorgerufen werden. In diesem Forschungsbezirk sind Schädigungen ganz verschiedenen Ursprunges in denselben Studien einbegriffen, als ob sie ein einziges Syndrom bildeten.

Eine grosse Forschungsarbeit muss unternommen werden. Der Geburtshelfer muss in seiner Klinik Forscher verschiedener anderer Fächer empfangen und leiten und muss ihre Untersuchungen, soweit die klinische Erfahrung die Hauptsache ist, beeinflussen.

REFERENCES

- Dawes, G. S., Mott, J. C. and Widdicombe, J. G. (1954) "The foetal circulation in the lamb." *J. Physiol. (Lond.)* **126**, 563.
- Dawes, G. S. (1957) Personal communication.
- Holland, E. (1922) *The Causation of Foetal Death*. Ministry of Health. Reports On Public Health and Medical Subjects, No. 7. London.
- James, L. S., Weisbrot, I. M., Prince, C. E., Holaday, D. A. and Apgar, V. (1958) "The acid-base status of human infants in relation to birth asphyxia and the onset of respiration." *J. Pediat.* **52**, 379.
- Lilienfield, A. M. and Pasamanick, B. (1955) "The association of maternal and fetal factors with the development of cerebral palsy and epilepsy." *Amer. J. Obstet. Gynec.* **70**, 93.
- Minkowski, A., Caillebotte, N., Saint-Anne-Dargassies, S., Larroche, J. C., Barab, R. and Maille, G. (1953) "La saturation en oxygène du sang artériel foetal à la naissance dans 158 cas." *Et. néo-natal.* **2**, 197.
- Mitchell, R. G. (1959) "Clinical aspects of a comprehensive survey of cerebral palsy." *Cerebral Palsy Bull.* **1**, No. 7, 32.
- Polani, P. E. (1958) "Prematurity and 'cerebral palsy'." *Brit. med. J.*, **ii**, 1497.
- Polani, P. E. (1959) "Nomenclature and classification." *Cerebral Palsy Bull.* No. 5, 53.
- Van Syke, D. (1959) Discussion on cord blood oxygen levels. In *Oxygen Supply to the Human Foetus. C.I.O.M.S. Symposium*. Edited by J. Walker and A. C. Turnbull. Oxford: Blackwell.
- Walker, J. (1955-56) "The oxygen environment of the human foetus." *Lectures on the Scientific Basis of Medicine*, Vol. V, 67. London: Athlone Press.
- Weisbrot, I. M., James, L. S., Prince, C. E., Holaday, D. A. and Apgar, V. (1958) "Acid-base homeostatis of the newborn infant during the first 24 hours of life." *J. Pediat.*, **52**, 395.

OBSTETRICAL FEATURES RELATED TO CEREBRAL PALSY

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So, the stout foetus, kicking and alive
Leaps from the fundus for his final dive.
Tired of the prison where his legs were curled
He pants, like Rasselas, for a wider world.
No more to him their wonted joys afford
The fringed placenta and the knotted cord.

Oliver Wendell Holmes.

Anoxia is the main cause of foetal and neonatal deaths, and infants who survive severe foetal anoxia are likely to be born defective or to develop defects in their early years. Foetal anoxia is largely a result of placental insufficiency, which cannot yet be accurately estimated in utero. The foetus is normally short of oxygen in the last weeks of pregnancy and becomes dangerously so if pregnancy continues into the 42nd or 43rd week. To reduce this danger, labour has been induced by rupture of the forewaters 5 days after the expected date of delivery in 2770 cases. The results seem promising.

Anoxia.—“Here then is Mount Everest in utero. Here are the same sparse oxygen pressures and the same phenomena of acclimatization occurring alike in the foetus and on the heights of the Himalayas. Here is Mount Everest and more, because in utero, as a result of certain pathological states, the already scant oxygen supply is sometimes reduced even further to levels which kill or maim untold myriads of infants.” (Eastman, 1954).

Physiology.—In utero the foetus exists at an oxygen environment corresponding to a level of 33,000 feet—“three-quarters of a mile above the summit of Mount Everest” (Eastman, 1954). Mountain climbers and those of us with Air Force experience know that above 20,000 feet the individual (who may not believe it to be so on his return to normal levels!) suffers from mental aberrations and altered behaviour. Unless protected by an artificial oxygen supply, the same indivi-

dual from about 14,000 feet upwards will experience a progressive deterioration of voluntary muscular control and co-ordination. “The principle cause of death before birth or in the first few weeks of extra-uterine life is interference with oxygenation. If anoxia were to be given as the cause of death of every foetus and young infant it would be correct as the immediate cause in almost all instances” (Potter, 1952). But not all the babies die when affected by anoxia, and it may not be till the second or third year of life, when speech and locomotion are developed, that defects become apparent.

“Oh, the little more, and how much it is!
And the little less, and what worlds away!”
Robert Browning: *By the Fireside*

Increased marrow activity and an average haemoglobin concentration at birth of 17 g./100 ml. show the degree of

acclimatization developed by the foetus in utero. But why does the foetus have to acclimatize? In all placental mammals, because of diffusion gradients, foetal tissues must necessarily exist under an oxygen pressure decidedly lower than that of the maternal tissues. The diffusion into the intervillous spaces of the placenta and further through the layers of the chorionic villi is such that in the human being the oxygen pressure in the foetal arterial blood is one-third that of adult blood.

Pathology.—The pathology of anoxia may therefore be said to be largely that of placental insufficiency. Browne and Veall, in 1953, showed conclusively that "The normal maternal placental blood-flow between 38 weeks and term is of the order of 600 ml./min., and in pre-eclampsia and chronic hypertension there is a reduction to about one-third of this figure." and "Because this reduction occurs in chronic hypertensive as well as pre-eclamptic women, it is suggested that the origin of the hypertension is extra-placental, though this does not exclude a further rise due to placental ischaemia." and "The healthy placenta has a functional safety margin of over 50 per cent. It is also inferred that the marginal sinus does not function as a distal collecting vein for the maternal blood leaving the placenta, each cotyledon having probably its own arterial supply and venous drainage."

There are other causes of placental insufficiency. Feeney (1954) in his "Obstetrical Alphabet" has classified the causes of placental insufficiency as follows:

(1) "Habitual: it occasionally happens that each successive foetus born to a mother is small, but well-nourished and normally developed. The placenta, too, is undersized, but well-formed and apparently normal on naked-eye and microscopical examination. It may be that the small placenta is responsible for the small foetus or that the retarded growth of the foetus and placenta occurs *pari passu* for some other reason."

(2) "Hormonal: if the development and implantation of the placenta depend to a great extent upon oestrogen and progesterone, it is reasonable to assume that hormonal deficiency

or dysfunction would result in placental insufficiency. Thus, oestrogens and progesterone are commonly employed for the repeated abortion and miscarriage case, but the results are not always satisfactory."

(3) "Faults in the structure of the placenta include placenta membranacea, circumvallata and accreta, in all of which the vitality of the foetus may be affected."

(4) "Faults in the location of the placenta include placenta praevia and implantation upon scars, fibroids and congenital septa."

(5) "Toxaemia of pregnancy; in this common condition, the serious effects of vasospasm, arteriolar degeneration, haemorrhage, ischaemic necrosis and infarction of the placenta, with or without premature separation, are well known."

(6) "Fibrinogen deficiency: giving rise to haemorrhage at the utero-placental junction."

(7) "Toxic accidental haemorrhage, other than of toxæmic origin, occurring as a result of hypertension or vascular disruption of unknown cause."

(8) "Non-toxic accidental haemorrhage due to blows, kicks or falls and external or internal version: following rapid evacuation of the liquor in hydramnios or shrinkage of the placental site after delivery of a first twin; due to premature separation of unknown origin."

(9) "Syphilis, in which the placenta may be large, but insufficient in function."

(10) "Tuberculosis and other systemic infections which are capable of affecting the placenta."

(11) "Vesicular degeneration of the chorionic villi, leading usually to the death of the ovum in the early months."

(12) "Cystic and other varieties of degeneration of the formed placenta, of doubtful origin."

(13) "Neoplastic changes, including chorio-angioma and metastatic deposits."

(14) "Spasm of the myometrium leading to occlusion of the utero-placental arterioles and induced by posterior pituitary extract or other powerful oxytocic substance, or occurring in the course of hypertonic inco-ordinate action."

(15) "Postmaturity and premature senility of the placenta with villous degeneration, vascular coagulation, fibrosis or calcification."

(16) "Anaesthetic complications with de-oxygenation, giving rise to faulty placental gaseous exchange."

(17) (The omission of diabetes as a cause of placental insufficiency is noted but not condoned).

I am not going to comment on all these causes, but it will be useful to consider several of them.

Threatened Miscarriage.—In threatened miscarriage in the 3rd or 4th month soon after the formation of the placenta it is reasonable to postulate that, because of the haemorrhage, there has been some

degree of placental separation which may reduce placental function, and in such a case it would be wise to induce labour a week or two before term. This was done in about 3% of the cases in the 5-year clinical review reported later.

Analgesia and Anaesthesia.—The question of analgesia or anaesthesia during labour requires careful analysis, especially with reference to its effects on the baby. This is highly pertinent at a time when the dangers of anaesthesia in pregnancy and labour are giving widespread concern because of the maternal deaths due to inhaling vomit during administration (Parker, 1945).

Postmaturity.—Walker (1953) has shown that "The per-cent saturation of the blood in the umbilical vein in the latter half of normal pregnancy is 70% at or about the 30th week, 60% at 39-40 weeks, and falls to under 30% at 43 weeks. All babies are not, however, even as well oxygenated as this and the blood of some infants in apparently normal pregnancy is only 30-40% saturated even at 40 weeks. A deficient oxygen supply may therefore be seen at any time in the last few weeks of pregnancy but by the 42-43rd week the supply is dangerously low in almost all foetuses." He says:

"(1) Induce labour before 40 weeks in cases of pre-eclampsia, or in those in which bleeding has occurred in the early months of pregnancy. (2) Primigravidae under 25 with otherwise normal pregnancies are allowed to go into labour spontaneously, but induction is performed if, after term, maternal weight is falling steadily, the liquor appears to be lessening, or pre-eclampsia develops. (3) If foetal distress develops during labour, and especially if meconium is present in the liquor, oxygen is given to the mother and the case assessed on clinical grounds. Especially in the older primigravida, and if a further period of prolonged labour is to be expected, caesarean section is seriously considered on the indication of 'foetal distress'. With the already anoxic foetus, caesarean section is often preferred if forceps delivery would be difficult and might put an undue strain on the foetus. (4) In primigravidae over 25 labour is induced at the end of the 41st week. If, at induction, the liquor is heavily stained with meconium, elective caesarean section is performed."

Mackay (1957) has confirmed Walker's findings, but Bancroft-Livingstone and Neill (1957) do not agree with them.

Trauma

In 1920 Holland read his now classic paper to the Edinburgh Obstetrical Society on "Cranial stress in the foetus during labour and on the effects of excessive stress on the intracranial contents; with an analysis of eighty-one cases of torn tentorium cerebelli and subdural cerebral haemorrhage". He reported that "out of 168 fresh foetuses the tentorium cerebelli was found torn in 81 (48%), associated with tearing of the falx cerebri in 5 cases and with subdural cerebral haemorrhage in all but 6. Most were the results of breech or forceps delivery, or of delivery through a contracted pelvis, but a few resulted from apparently normal labour". I would commend a re-reading of this paper to all obstetricians especially to the younger ones. Perhaps its greatest lesson is that one should closely follow the natural mechanism of labour in all operative deliveries—something perhaps not properly appreciated by the younger obstetrician because of the recent great improvement in the size and shape of the female pelvis as a result of improved diet and prevention of rickets. When I re-read the paper I also read the subsequent discussion and I crave indulgence to quote at some length what Dr. Ballantyne said. (How often we now return to Dr. Ballantyne, the instigator of pre-natal care!) He (Dr. Ballantyne) then spoke of the causes of cerebral haemorrhage:

"He thought that to have the idea that cerebral haemorrhages were anything like all of them due to the tentorial injuries and the resulting injuries to the veins—the veins of Galen and the internal cerebral veins, etc.—would be to throw the emphasis too strongly upon that particular cause. He and Dr. Browne (F. J.) had often found, with no interference at all and after easy labours, very considerable intracranial haemorrhages and the explanation which had occurred to them was prematurity. " . . . He wondered if, in the case of children who do not die in actual birth but die some weeks later, the intracranial haemorrhage is

not due to asphyxia. Another question was whether these haemorrhages were the result of postnatal infection. Dr. Ballantyne mentioned a paper contributed to a journal by Sir William Osler on intracerebral haemorrhage in a foetus of six months, where the mother had died. The foetus was extracted from the abdomen and intracranial haemorrhage was found, showing that a foetus may have intracerebral haemorrhage which occurs before and which is quite independent of the mechanism of labour. That was the first point to be kept in mind—that these haemorrhages may be caused before birth. The second cause was haemorrhage during the strain of birth. The third cause was the child being born into a new environment laden with germs, and infection coming in."

Today we are often still in the dark as to whether asphyxia causes haemorrhage or haemorrhage causes asphyxia.

Clinical Applications of New Knowledge

In Bristol we have followed Walker's recommendations with regard to post-maturity, and indeed in the past 5 years over 30% of the cases of induction have been because of postmaturity. This has meant that labour has been induced by rupture of the forewaters 5 days after the expected date of delivery in the hope that the baby would be out of the uterus before the end of the 41st week of pregnancy. At the end of the first year (1954) of this clinical research the results were so good, especially from the perinatal mortality point of view, that it was decided to continue the method, and now after 5 years 2770 inductions by rupture of the forewaters have been carried out. This has given an induction-rate of 40.3% of cases in the Professorial Unit at Southmead Hospital, Bristol, and the inductions were done for the following reasons:

Pre-eclampsia and other forms of hypertension	47.8%
Postmaturity	33.4%
All other indications	18.8%
(Total deliveries over 5-year period 6881)	

The dangers to the mother, as far as we could see, were as follows:

Prolonged induction-delivery interval (over 48 hours) ...	20%
Infection	1.4%
Prolapse of cord	0.4%
Prolonged 1st stage of labour (over 24 hours)	3.5%

One mother died of hypertension due to pyelonephritis: seven years previously she had been advised never to become pregnant again.

It is obvious that many needless inductions of labour are carried out by this method, but it is very difficult to select the proper cases for induction—that is, those who would get foetal distress during labour and/or stillbirth or cerebral anoxia. Our induction figures have been comprehensive rather than particular. There is no doubt that this programme of induction of labour by rupture of the forewaters and the release of liquor amnii has led to quicker labours, by shortening the first stage. This does not mean an increase in the caesarean section rate because of those cases that failed to go into labour. In 11.5% of cases an oxytocic drip was used in conjunction with rupture of the forewaters and then labour ensued. One of the interesting points is that there have been few case of really difficult forceps delivery—uterine action seems to have improved and consequently so has the mechanism of labour.

The results so far as the foetus is concerned have been as follows:

Total number of inductions ...	2770
Stillbirths	61
Neonatal deaths	18

This gives an uncorrected rate for stillbirths and neonatal deaths of 2.85%.

Out of 79 babies lost, 24 were grossly abnormal.

Of the 55 normal babies, 18 weighed under 4 lb.

Of the deaths, 41.8% were after induction for pre-eclampsia or hypertension; 8.9% were after induction for post-maturity.

The oxytocic drip method has been criticised, but our results, supporting Theobald (1956), are as follows:

Total cases in which oxytocic drip was used	318
Cases ending in loss of the baby	24
Gross congenital defect	10
Gestation under 28 weeks	2
Babies under 4 lb. weight	9
(including 2 under 28 weeks).	
<i>Deaths related to Indications for Induction</i>	
Pre-eclampsia, hypertension and one eclampsia	9
Postmaturity	2
Foetal abnormality	10
Accidental haemorrhage	3
<i>Overall Caesarean Section Rate...</i>	2.4%
<i>Forceps-rate (including prophylactic forceps delivery in pre-eclampsia and foetal distress)</i>	13.5%

Comment

Until an easy method is devised for estimating the degree of placental insufficiency, particularly in cases of pre-eclampsia and postmaturity, induction of labour by rupture of the forewaters \pm oxytocic drip offers in our hands a method of delivery which is safe for the mother and safer for the baby than the hazards of placental insufficiency due to pre-eclampsia, hypertension and postmaturity. "Leaving it to nature" does not produce comparably good figures. But leaving statistics aside, the good results recorded above have been achieved easily and without the worries of prolonged labour and difficult forceps delivery. The same results can be achieved in two ways—the hard way with "blood, and sweat, and toil" or the easy way described here. Who dares contest that easier and quicker birth, be-

fore anoxia can have any effect, must prevent brain damage and the unpleasant sequela of cerebral palsy?

Research Needed

A great deal remains to be done on this subject. A little has been done in retrospect by the paediatrician, but the obstetrician is in the position to undertake more research on the protection of the nervous system during pregnancy and labour. For example, a more reliable method is needed for estimating placental insufficiency. Radioactive isotopes have been used for this but have their own dangers and have not given the results expected. The obstetrician should pay particular attention to the carbon-dioxide tension and the effects of maternal sedation on the baby during the administration of analgesic drugs and anaesthesia.

Whether the practice described above, of increasing the number of inductions of premature labour, is as good as it seems requires further investigation, especially with regard to whether there are more spastic children from such cases when labour has not been accompanied by the release of liquor amnii. The causes of prematurity and the damage done to the nervous system by early release from the uterus should also be investigated.

A cure for pre-eclampsia, the cause of which still remains obscure, must be found. The subject cries out for research, and the co-operation of paediatrician, anaesthetist and obstetrician should be close. Indeed, the programme from this point of view should be co-ordinated.

SUMMARY

The physiology and pathology of foetal anoxia are considered, and the part played by placental insufficiency, particularly in toxæmia and postmaturity, is reviewed in the light of present-day knowledge.

Further studies are needed on foetal oxygen depletion, for example secondary

to analgesia, anaesthesia and cerebral birth trauma.

An analysis of 2,770 inductions of labour by artificial rupture of the forewaters has been made, and attention is drawn to the prophylactic value of the procedure and its importance in foetal salvage.

RESUME

Caractères obstétricaux concernant l'infirmité motrice cérébrale.

Après avoir considéré la physiologie et l'anatomo-pathologie de l'anoxie foetale, le rôle joué par l'insuffisance placentaire, en particulier dans la toxémie et la post-maturité, est passé en revue à la lumière des connaissances actuelles.

De nouvelles recherches s'imposent et doivent porter sur la déplétion en oxygène

du fœtus, par exemple, consécutive à l'analgésie, l'anesthésie et au traumatisme obstétrical cérébral.

2770 cas de travail provoqué par rupture artificielle des eaux ont été analysés. L'attention a été attirée sur la valeur prophylactique de ce procédé et de son importance sur la protection du fœtus.

ZUSAMMENFASSUNG

Geburtshilfliche Beschaffenheiten der zerebralen Kinderlähmung.

Nachdem die Physiologie und die anatomische Pathologie der fötalen Anoxämie betrachtet wurden, wird die Rolle der Mangelhaftigkeit des Plazentas, besonders bei Toxämie und Überreife, in Hinblick auf die jetzigen Kenntnisse, untersucht.

Neue Forschungen sind dringlich nötig und müssen auf den Sauerstoffmangel der Frucht, zum Beispiel während der Analgesie,

der Anästhesie und des zerebralen Traumas der Geburt gerichtet sein.

2770 Fälle von durch Eihautstich hervorgerufenen Entbindungen wurden untersucht. Es wurde auf den prophylaktischen Wert dieses Verfahrens und auf dessen Wichtigkeit für die Beschützung der Frucht hingewiesen.

REFERENCES

- Ballantyne, J. W. (1920) Discussion on paper of Holland (1920). *Trans. Edinb. obstet. Soc.*, **40**, 138.
- Bancroft-Livingstone, G. and Neill, D. W. (1957) "Studies in prolonged pregnancy. Part I—Cord blood oxygen levels at delivery." *J. Obstet. Gynaec. Brit. Emp.*, **64**, 498.
- Browne, J. C. M. and Veall, N. (1953) "The maternal placental blood flow in normotensive and hypertensive women." *Ibid.*, **60**, 141.
- Eastman, N. J. (1954) "Mount Everest in utero." *Amer. J. Obst. Gynec.*, **67**, 701.
- Feeney, J. K. (1954) "An obstetrical alphabet." *J. Irish med. Ass.*, **34**, 118; 150; 190.
- Holland, E. (1920) "On cranial stress in the foetus during labour and on the effects of excessive stress on the intracranial contents; with an analysis of 81 cases of torn tentorium cerebelli and subdural cerebral haemorrhage." *Trans. Edinb. obstet. Soc.*, **40**, 112.
- MacKay, R. B. (1957) "Observations on the oxygenation of the foetus in normal and abnormal pregnancy." *J. Obstet. Gynaec. Brit. Emp.*, **64**, 185.
- Parker, R. B. (1954) "Risk from aspiration of vomit during obstetric anaesthesia." *Brit. Med. J.*, **2**, 65.
- Potter, E. L. (1952, Pathology of the Fetus and the Newborn, p. 54. Chicago: Year Book Publishers.
- Walker, J. (1954) "Foetal anoxia" *J. Obstet. Gynaec. Brit. Emp.*, **61**, 162.

Comments On The Papers Of Prof. James Walker and Prof. G. Gordon Lennon

COMMENT NUMBER I.

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WHEN William Harvey (1578-1657) described the placenta as the "uterine liver" and John Mayow (1643-1679) later described it as the "uterine lung", they may or may not have appreciated something of the enormous prospective significance of their beliefs. Harvey was deeply interested in the foetus and its birth. He posed many questions. He said: "in labour it often happens that the foetus is retained many hours without the possibility of breathing yet is found to be alive; when, however, it is once born and has breathed, if you deprive it of air it dies at once". Also, "why should the foetus remain in utero after the seventh month, because if born at this time not only does it breathe but without respiration cannot live one little hour, yet, if it remains in utero it lives in health and vigour more than two months longer without the aid of respiration at all?"

Ten years after Harvey's death (1657) Mayow cleared up some of Harvey's difficulties by postulating that the placenta was a form of lung. In fact, Mayow showed that only a part of the air was used in burning and in respiration: the nitro-aerial or the igneo-aerial particles were "essential for all the chemical changes on which life depends". Mayow asked (rather similarly to Harvey) "how it happens that the foetus can live through imprisonment in the straits of the womb and completely destitute of air?". He answered it by the brilliant statement "that the blood of the embryo (foetus), conveyed

by the umbilical arteries to the placenta, brings not only nutritious juice, but along with this a portion of nitro-aerial particles by its circulation through the umbilical vessels quite in the same way as in the pulmonary vessels, and therefore, I think, that the placenta should be called a uterine lung".

In 1772 Joseph Priestley isolated oxygen. Claude Bernard (1813-1878) later found glycogen in the placenta; thus Harvey's idea of uterine "liver" and Mayow's suggestion of uterine "lung" were unified.

It is remarkable that, concurrent with the advances recorded by Mayow, the following was a general belief: "For sharp and difficult travail in women with child take a lock of virgins haire on any part of ye head, of half the age of ye woman in travill. Cut it very smale to fine powder then take 12 ants egg dried in an oven after ye breade is drawne or otherwise make them dry and make them to powder with the haire, give this with a quarter of a pint of red cow's milk or for want of it give it in strong ale wort".

Beliefs were difficult to eradicate. The fantastic "Byrthe Fygue" in Eucharius Rösslin's "De Partu Hominis" (1535), including the "flying foetus" and the modes of escape from the uterus, were for many years authoritative. The dissections of the gravid uterus by William Smellie (1754) and the publication of William Hunter's "The Anatomy of the Human Gravid

Uterus" (1774) revealed at last the real dispositions and attitudes of the foetus in utero and (as shown in No. 8 of William Hunter's plates) the placement of the placenta and the umbilical cord.

Hitherto, foetal attitudes had existed only in the imagination and fantasy. Smellie continued in the Harveian tradition and found out the facts by way of personal observation and experiment. The "secrets" of the foetal environment were now known and a more rational understanding of the processes and mechanisms of birth soon followed. William Hunter and his brother, John Hunter, described the anatomy of the symphysis pubis in pregnant women. It is likely that John rather than William first discovered the independence of the circulation of the foetus and the mother, and thus made the first significant contribution to our knowledge of the anatomy of the placenta.

"As a teacher, author and practitioner, there is no British obstetrician—certainly none of the eighteenth century—who deserves so high a place as William Smellie": we agree. "A Treatise on the Theory and Practice of Midwifery" (1752) records his famous work. The proper study of midwifery, even to-day, best begins at that point of time in the essays: "Of Natural Labours", and "Of Laborious Labours", and "Of Preternatural Labours", and "Of Lingering or Tedious Labours", each of which is utterly convincing. This great clinician leaves us also in no doubt about his anxieties for the foetus and the health or illness of the newborn. The perinatal period was commonly and certainly loaded with hazards. The "valley of the shadow of birth" was truly deep and dangerous.

This, no doubt, caused others to ponder on these matters, but we are principally indebted to W. J. Little for the logical follow-up: the publication of Little's paper in 1862, "On the influence of abnormal parturition, difficult labours, premature birth, and asphyxia neonatorum, on the mental and physical condition of the child", focused the urgent needs in regard to the greater safety and well-being of the foetus and the newborn. The intrapartum life came into acute focus—"there is an epoch of existence, viz. the period of birth, during

which we might consider that the foetal organism is subjected to conditions so different to those of its earlier and of its prospective later existence, that any untoward influences applied at this important juncture would affect the economy in a manner different to the influences at work during the periods ordinarily characterised as those of before onset of birth and after birth"; and, moreover "reflection on the nature of delay in the substitution of pulmonary for the ceased placental respiration would lead to the apprehension that the want may imprint a lasting injury upon the new-born child".

It is interesting that Little was addressing his remarks to the Obstetrical Society of London. He himself was senior physician to the London Hospital. The obstetricians agreed that Dr. Little "had brought before the obstetric world new matters for inquiry of the highest interest; he had pointed out the injuries to which the nervous system was liable during, and immediately after, birth". Notions about postnatal paralyses detected later were still linked with "the irritation of teething" and even such statements as "reflex paraplegia ensued during dentition".

Little struggled against these dangerous vagaries of teleology. The carry-over from birth seemed, to many, quite occult—"these forms of paralysis (now called cerebral palsy) were commonly met with from six months of age". However, light was brought to the matter by a report in *The Lancet* of Nov. 13, 1858, in which "after a lingering labour, a child was born with spastic rigidity of all the muscles on one side of the body . . . in fact a conjunctive (sic) hemiplegia".

Greater obstetrical interest was stimulated by Little's oration: Dr. Barnes "hoped that the study of the causes of still-birth would be more closely prosecuted by post-mortem examinations", and the same speaker, on searching his mind, observed that Samuel Johnson* "was born (1709) almost dead, and did not cry for some time; and although his name was almost synonymous with intellectual grandeur he was well known to be affected with certain nervous

*He was a tiqueur. Ed. *Cerebral Palsy Bulletin*.

disorders which Dr. Little could better interpret". Dr. Barnes confessed to "not having studied the subsequent history of children in connexion with the phenomena attending their birth".

An enormous step forward occurred in 1901, when J. W. Ballantyne made his "Plea for a Pre-Maternity Hospital". At this time Ballantyne actually held a post entitled Lecturer in Antenatal Pathology in the University of Edinburgh. This was certainly far sighted. He wished to develop also a hospital where antenatal disease and its possible consequences to the foetus could be scientifically investigated. He had already noted that working women who would rest for the last few weeks of pregnancy gave birth to "healthier" infants. Ballantyne was somewhat pessimistic about his being able to influence hospital authorities or perhaps even some of his colleagues, even confessing that "the idea will be regarded as visionary or chimerical" and that it had been only by slow degrees that he had come to regard it as anything else!

The problem of the post-mature infant occupied Ballantyne's special attention. His comparative studies caused him to state categorically that while the premature infant "can without difficulty be born alive . . . the immature is face to face with the difficulty of behaving in all respects as a newborn when he is yet only a foetus: his troubles begin after birth . . . the postmature infant, on the other hand, has stayed too long in intra-uterine surroundings; his troubles come on during birth . . . the problem of the postmature is intranatal". And: "To be expelled timeously from the uterus is almost as important as to be nourished perfectly in it . . . the partus praecox is one thing and the partus serotinus another, . . . degeneration of the syncytial layer and other signs of placental deterioration are important factors . . . the management of the post-mature infant that survives his birth cannot be said to be well-understood . . . the chief treatment will require to be directed towards undoing the evil effects of birth traumatism.

The period 1900-1950—so called "The Harvest" because of the quickening of obstetric medicine by the scientific method and advancing clinical and laboratory

sciences, so "unparalleled in its bounteousness"—has advanced our knowledge on embryology; the innervation and actions of the uterus; sex physiology; pregnancy; labour; complications of labour; pharmacology, especially of oxytocic drugs; "toxaemia" of pregnancy; obstetric anaesthesia and analgesia; radiology in obstetrics; and other matters, not least, the social, economic, nutritional and environmental factors for or against maternal health and foetal welfare. In the middle of this period (1923) Ballantyne (in probably the last written paper of his life) wrote on "The New Midwifery" and therein he made clear his mind:

"In the 'old', prevention and repair were attempted practically within the time limits of the labour itself whereas in the 'new' prevention begins away back in the early months of pregnancy, if not earlier still, and reparative measures pursue the patient without regard to any time limit and solely with respect to complete restoration of the damaged parts to their original structural and functional efficacy. In the past it has been far too common for the medical attendant to concentrate his whole attention upon the supremely critical hours in which the child is passing through the mother's pelvis in the act of birth; it has been his pride, and also his sole endeavour, to step promptly forward when labour has been evidently not proceeding normally, to 'sense' in a flash what has been wrong, and brilliantly, by ingenious manual or instrumental or operative devices, to correct what has been amiss, presenting triumphantly to an impressive father and a relieved mother a more or less uninjured baby. At the end of a fortnight or three weeks he has been in the habit of saying farewell to his patients after a somewhat perfunctory inquiry into their fitness. By preventive measures founded upon ante-natal supervision and treatment the obstetrician of the present, and still more of the future, is and will be able to gain a less dramatic, a less spectacular, but an infinitely more comfortable and less exacting victory in labour; and by a careful and continued 'follow-up' of his patients (mother and infant) he will be enabled to return them to the ranks, so to say, of the general population intact and fit for what the future may demand of them".

"The new midwifery with its provisions will at once lessen the still-births rate. Statistics from other places show something like a 50 per cent reduction. I was able to produce figures indicating a rate of 13.5 per 1,000 among 816 ante-natally supervised pregnancies, including over 100 in which there was the complication of one or other or both the venereal diseases. These cases occurred in the ante-natal department in the Edinburgh Royal Maternity Hospital during 1921. The rate for the whole of Edinburgh was 47.8. If the venereal cases were excluded the still-births rate fell to 5.9 per 1,000".

By 1950* the preventive aspects were clearer to all. It seemed certain that a more general application of present day knowledge of obstetrics and paediatrics by the medical and nursing professions and by those responsible for social services and for administering hospital and domiciliary midwifery services would result in a further considerable reduction in the neonatal mortality rate. Similarly, if every pregnant woman received first-class medical and nursing supervision during pregnancy and labour, the still-birth rate also could be appreciably decreased. It is abundantly clear that steps should be taken to stress the importance of the life and health of the newly born baby.

It is against this background that we read the articles of Prof. G. Gordon Lennon and Prof. James Walker in this issue. These writers are in line with principles hinted at by Smellie and Ballantyne, and they exemplify the great and increasing significance of the physiology of pregnancy and its related essentials at all levels in the mother, the embryo, the foetus and the newborn. Continuity of study and care from conception to puerperium and later is the keystone of the exercise. The obstetrician as a clinical physiologist is outlined by Lennon and by Walker: each has presented a picture in clear, concise and meaningful language with an expressed appreciation of the shifts away towards the unhealthy, the abnormal and on to the pathology; yet in both papers the fundamental importance of preventative aspects is kept clearly in focus. The forthright statements of fact and the clinical sense and awareness indicate, without any doubt whatever, the present-day hopes and achievements in and for foetal health, safer birth and post-natal care of the highest order. It is notable, also, that each writer consciously expresses an attitude of humanitarianism; an attachment of high professional knowledge to a practical application

of skills in an overall understanding of the needs, urgent and otherwise, in saving the foetus and the baby from embarrassment, stress, injury or "brain damage".

Obstetrics lends itself to two principles: (a) unflinching vigilance; (b) dangers and risks inherent in reducing obstetric responsibility to a banal routine. First-class obstetrics is first-class clinical preventive medicine: non-meddlesome obstetrics is a strong contribution to preventive gynaecology. The continuum of reproductive casualty will be kept at its lowest minimum by the understanding and wisdom expressed by our two obstetricians. Each has written quite independently—one prefers "obstetrical features" and the other prefers "obstetrical viewpoints"—but both write with a close and diligent understanding of cerebral palsy and how present-day obstetric practice should be focused on such an enormously important and possibly, in a high proportion of cases, preventable syndrome. More exact knowledge concerning the aetiology of cerebral palsy will probably become available as a result of some of the present organised prospective research: such is an urgent necessity for forward progress. Obstetrical factors in the aetiology of handicapped children must be more clearly understood. In considering "The Future", James Walker poses the statement: "Perhaps our standards are not high enough . . . how many children are damaged, just a little," (my italics) "so that they are well below their potential level but not obviously defective?"—how reminiscent of W. J. Little! Gordon Lennon is equally helpful—"the obstetrician is in the position to undertake more research on the protection of the nervous system during pregnancy and labour . . . the coöperation of paediatrician, anaesthetist and obstetrician should be close". The needs are clear: Birth without foetal, or perinatal, or neonatal or any other postnatal morbidity of the nervous system.

*See Rep. publ. Hlth. med. Subj. No. 94. "Neonatal Mortality and Morbidity". London: H.M. Stationery Office, 1950.

COMMENT NUMBER II

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The contributions of Prof. Gordon Lennon and Prof. James Walker are rich and provocative and I concur wholeheartedly with most of what both authors write.

For the convenience of the reader I will attempt, where possible, to annotate longitudinally and separately. To begin with Professor Lennon's contribution, I would prefer to modify his statement on physiology to read that defects are best sought during the first year of life rather than in the second or third. The paediatrician well trained in child development can make the diagnosis of moderate or severe defect quite as readily during the first year as later. In addition the evidence of minimal cerebral damage frequently disappears or is compensated for in succeeding years and is therefore missed. There is a good deal of evidence that this type of injury may contribute to behavioral and learning defects later in childhood.

In discussing pathology, consideration should be given to the possibility, for which preliminary data exist, that anoxic or other damage may be incurred by the foetus before clinical signs of toxæmia develop in the mother. Both maternal toxæmia and foetal damage may follow events occurring as early as the first trimester.

I am intrigued by Professor Lennon's suggestions and data on induction of labour for postmaturity and maternal hypertension. He is well aware of the dangers of prematurity, infection, prolonged labour etc. The perinatal mortality rates quoted are so

low that the immediate institution of an experimental study to test definitively the clinical impressions would be highly advisable. Professor Lennon is undoubtedly aware that in an excellent obstetric service such as his factors other than induction of labour might have contributed to the decreased mortality. In addition, perinatal mortality is not the only and possibly not even the best index of obstetric care. Other constituents of the continuum of reproductive casualty must also be considered. Spastic children could be only one of the indices because of their relatively low incidence. Other sequelae of brain damage must be sought and measured. A well-designed prospective study, with patients randomly assigned to the group with the experimental independent variable of elective induction of labour, seems to be most urgently required.

Our debt to Dr. Ballantyne appears endless. He is indeed the father of us all. I was not acquainted with the passage quoted by Professor Lennon but am not at all surprised at its existence since Dr. Ballantyne seems to have anticipated almost all our findings and theories in his Antenatal Pathology and other writings.

In our own Baltimore retrospective and prospective studies we encountered no measurable effect of operative and difficult labours. We were led to conclude, as does Professor Walker, that this was largely due to advances in obstetric practice. Some more recent direct pathological evidence leads us to believe that it is the foetal or neonatal

brain predisposed by maternal complications and prematurity that may be damaged by mechanical factors. The experimental data derived from animals tend to confirm the impression that asphyxia may produce haemorrhage.

To turn now to Professor Walker's contribution I would like to subscribe wholeheartedly to the succinct and felicitously phrased introduction to his paper. The need for additional effort by the obstetrician and gynaecologist and others is imperative.

Professor Walker's paper includes two broad areas to which I would like to add my comments—viz., socio-economic versus genetic factors and research design in elucidating aetiological variables. For example, he points to short stature as being associated with obstetric difficulties and birth trauma. However, short stature is also associated with other complications of pregnancy and labour, such as toxæmias, bleeding, prematurity, heart disease, and infection. Short stature, probably through these precursors and others, is also associated with increased risk of foetal and infant death and brain damaged offspring. Low socio-economic status is in turn, and probably causatively, associated with short stature.

A host of studies in the last few decades, including the now classical investigations of Franz Boaz, the anthropologist, and the British experiences with the improvement of status, indicate quite strongly that it is post-conceptual life experiences which largely determine stature and the variables associated with it. It is the unfortunate spiral, consisting of noxious environment, including under- and mal-nutrition and stress of all types, associated with poverty and deprivation throughout life, that leads to brain damage and lowered intellectual capacity, which in turn lead to further lowering of socio-economic status and all the concomitants enumerated above. It is this conceptual model rather than the genetic model which scientific parsimony and the weight of the evidence would demand.

I cannot therefore agree with Professor Walker's statement that mental deficiency is probably highly correlated with heredity and only rarely with pregnancy and labour, or that certain community groups (apparently

those in lower economic strata) have a poor genetic background. The only evidence advanced for the genetic model is the post hoc one of familial aggregation. However, familial aggregation of all the constituents of the spiral described previously is at least as good, if not a better, explanation for the occurrence of mental deficiency and other forms of brain damage, particularly in view of the repeated observations that economic improvement is associated with marked changes for the better in stature and functioning within one or two generations—obviously too short a time for genetic variables to play a role.

Professor Walker makes a series of comments under the heading of "Obstetrical Factors in the Aetiology of Handicapped Children" with which I cannot wholly agree. His first statement is that retrospective studies of pregnancy and labour are almost bound to fail because of inaccurate or incomplete records. There is little doubt that most hospital records are incomplete and even intentionally distorted when some member of the obstetric team may lay himself open to criticism. It should be noted here that the danger of incomplete records lies wholly in the possibility that no associations may be demonstrated—in other words, that bias may exist against the hypothesis. However, when positive associations are found it only means that the associations are probably even stronger than the data indicate, since in a well-controlled investigation there is no reason to believe that there should be differential recording of data for experimental and control populations.

Professor Walker also complains that in field studies conditions such as cerebral palsy, epilepsy, behaviour defect and mental deficiency are lumped together even though aetiological causes vary widely. Further, that these are not single conditions. While these observations are valid this does not negate the possibility of epidemiological investigation. Again the danger is one of a bias against the hypothesis. If a condition has many possible causes and the associations of that condition are sought with one or a similar group of conditions, the possibility exists that if comparatively small samples are used a positive association may be missed.

This is a distinct danger and certain precautions from the point of design and numbers must be taken. As every investigator knows, negative findings must always be scrutinized more carefully than positive findings. However, again, when positive associations are found under the conditions about which Professor Walker complains the association must be considered to be even more significant. The same observations may be made concerning the statement that the clinical entities mentioned are not single conditions.

The third point made is that diagnoses of the clinical conditions associated with brain damage have varying criteria as well as different levels of involvement. Again this may unfortunately be true, but it does not negate the possibility of instituting investigations. It is usually sufficient to specify the criteria, make certain that they are identical in experimental and control samples and, if desired, the data in both groups may be analyzed and compared criterion by criterion.

The last point made in this series is that the possibility of multiple aetiological factors may be present in the same case. Again this may be true. There are methods in which the responsibility of one factor can be estimated, controlling for the others and also for weighing the effect of a combination of variables. What is usually required is enough cases to permit assignment of these factors to individual cells and sufficient additional pertinent data to permit adjustment and control. Professor Walker pointed to the possibility that a number of aetiological factors may be due to a single cause. In effect, this tends to narrow the search to the more fundamental aetiological factor or factors.

Professor Walker proposes as a solution to these difficulties "a full-scale study of mothers (and fathers) during pregnancy and labour, with follow-up of all children". Unfortunately this type of prospective study is subject to all the difficulties previously mentioned, in addition to a few others. First the number of patients required to secure positive associations between cerebral palsy, a comparatively rare condition, or even between the more common conditions such as epilepsy or mental deficiency and the host

of pre-, para- and neo-natal events, simultaneously factoring out postnatal occurrences, would be forbiddingly enormous and expensive. To study the intercorrelations of a large number of multi-caused clinical conditions having varying levels of definition with an enormous number of events, singly and in combination, over a period of years using methods frequently unvalidated, is at present difficult to contemplate. Such studies, which could not be done by a single team of investigators, would require the achieving of comparability, reliability and validity of indices both independent and dependent; establishing and testing methods for these determinations before launching such an investigation. Finally, it must be obvious that no unforeseen associations could be found, since recording would cover only those factors already suspect or demonstrated by previous studies.

Some factors could not be investigated by this type of study at all. For instance, the effect of the induction of labour, previously discussed, could be determined only by an experimental study. In any event, the large prospective type of study, even if successfully completed, would not be definitive since only associations would be demonstrated and would require experimental verification for more definitive demonstration. There is also the danger that some efforts toward prevention might be postponed to await the results of the heroic, lengthy investigation.

I would instead propose a different type of study. This would consist of a single or series of experimental investigations in which attempts would be made to prevent one or more of the strongly suggestive factors in cerebral injury. Such factors as various dietary constituents instituted preconceptually, and the prevention of infection and other stresses could be suggested as independent variables. Dependent variables might be the constituents of the entire continuum of reproductive causality as well as measures of maternal morbidity. This would at one time permit definitive implication of fundamental variables, elucidate some of the mechanisms, and prevent the delay of the institution of preventive measures.

Professor Walker raises the question of

"How many children are damaged, just a little, so that they are well below their potential level but not obviously defective?" This is a most important question and one of which one of our investigations permits a preliminary estimate. Adjustment of the data from the Baltimore premature study would lead us to believe that the answer is no less than 10 per cent of the infant population.

I cannot fully comprehend the statement that "It is tempting to assume that prematurity and defective cerebral development are linked, but it is specifically this type of supposition that is incapable of proof without a great deal more study". Almost every well-designed study of prematurity has found a positive association with defective

cerebral development, and apart from an experimental study in which prematurity is deliberately provoked I am at a loss to think what further study is necessary to demonstrate the link. If it is the mechanisms of damage that require further study one can only agree. It would seem to me however that studies in the aetiology and prevention of prematurity would be even more desirable and would make the study of mechanisms of damage superfluous.

I would like to second most strongly Professor Walker's excellent suggestions of what the obstetrician can do immediately and to express my gratitude for this opportunity to comment on these most valuable contributions to the literature on cerebral palsy.

E.E.G. IN CEREBRAL PALSY

DR. MED. ARNE LUNDERVOLD.

From the University Neurological Clinic, Oslo.

This comparison of the clinical findings in a group of cerebral palsy patients with those obtained by air encephalography and encephalography demonstrates the importance of repeated E.E.G. examinations in establishing the prognosis and evolution of the lesion.

In our clinic at Oslo University the results of electroencephalography in cerebral palsy were at first somewhat disappointing. The correlation observed, for instance, between the E.E.G.s and the neurological and pneumoencephalographic findings in the clinically most severely affected group—the mixed one—was unsatisfactory, for 50 per cent of these patients had a normal E.E.G. The correlation between the seizure pattern in the E.E.G.s and the patients' clinical seizure was also poor, for 14 per cent of them had epileptic discharges in the E.E.G. with no epileptic seizures clinically. On the other hand, the correlation between the clinical, pneumoencephalographic and E.E.G. findings in patients with hemiparesis was extremely good. The E.E.G.s showed focal

disturbances, mostly in the form of a reduction in the normal voltage in the hemisphere contralateral to the hemiparesis. Similar good results were obtained in the epileptics, where the E.E.G.s showed seizure discharge in 90 per cent of the cases.

In a series of 223 patients examined by Dr. Skatvedt and myself the findings were as in tables I and II. The figures in parentheses represent patients who in addition to bilateral dysrhythmia showed a focal epileptogenic activity.

The pneumoencephalogram gave some similar results when compared with the neurological findings. The cerebral palsy patients with hemiparesis were the only group who gave a characteristic picture, and the only one for which the pneumoencepha-

Table I. Number of E.E.G. examinations done in various groups.

					Normal	Pathological	Total
Hemiparesis (right)	7	28	35
Hemiparesis (left)	1	19	20
Bilateral spasticity	37	35	72
Athetosis	15	20	35
Ataxia	5	10	15
Mixed group	23	23	46
All groups	88	135	223
					(39%)	(61%)	

Table II. Pathological E.E.G. findings in 135 patients with cerebral palsy.

	Focal dysrhythmias			General dysrhythmias		Bilateral synchronous spikes and/or spikes and waves
	Right side	Left side	Bilateral	severe-moderate	slight	
Hemiparesis (right)	—	22	2	4(3)	2	3(2)
Hemiparesis (left)	16	—	2	1(1)	1	1(1)
Bilateral spasticity	8	4	5	3(1)	12	4
Athetosis	1	6	1	1(1)	10	2
Ataxia	2	0	1	1	6	
Mixed group	4	3	4	10(5)	6	2(1)
All groups	31	35	15	20	37	12

The focal dysrhythmias consists partly of theta-delta waves, spikes, sharp waves, spikes and waves, or focal reduction of the normal cerebral electric activity, and partly of combinations of these forms.

Bilateral dysrhythmias include cases which partly show isolated epileptogenic foci in both hemispheres (spikes and spike-waves), and partly reduced cerebral activity or theta-delta activity in one hemisphere and epileptogenic foci in the other.

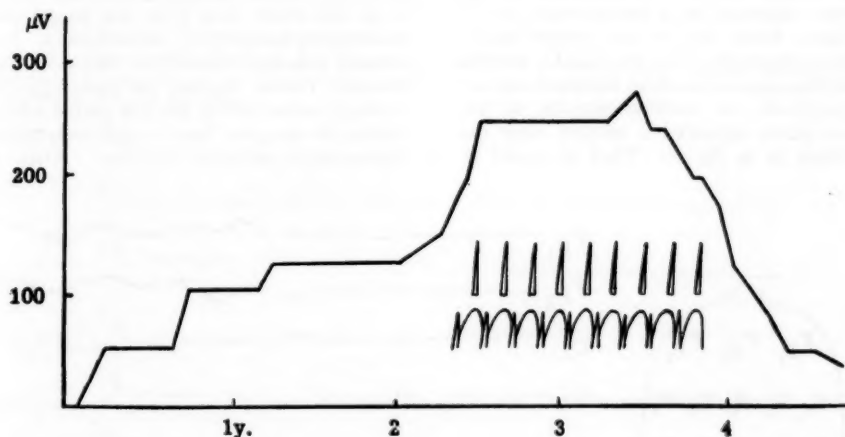


Fig. 1. Voltage of the slow waves in 33 EEG's recorded from the same patient in a period of 4½ years. The epileptiform activity is marked as spikes or spikes and waves. The patient had a severe brain injury 14 days before the first recording.

logram may suggest the probable diagnosis. In all other varieties of the disease the most common findings were a central dilatation of the ventricles. These results did not permit any conclusions to be drawn regarding the

clinical type of the disease. A correlation between the pneumoencephalogram and the electroencephalographic findings was satisfactory in the patients with hemiparesis. In one patient with right-sided hemiplegia,

however, the pneumoencephalogram was normal, but the E.E.G.s showed a marked focal dysrhythmia of the whole of the left hemisphere. This remarkable result will be explained later.

In about 33 per cent of the patients with a pathological pneumoencephalogram the E.E.G.s were normal, so the correlation as a whole between the E.E.G. and the pneumoencephalogram was not very good. Only one patient with epilepsy had a normal pneumoencephalogram.

A peculiarity in the electroencephalographic results which has also been observed by others, was that 14 per cent of the patients with cerebral palsy showed seizure discharge without having had any clinical epileptic seizures. Fig. 1, represents 33 E.E.G.s recorded from a patient over a period of $4\frac{1}{2}$ years, may explain or suggest an explanation for these.

A boy of 13 years accidentally had his larynx obstructed by a foreign body for 15 minutes. After that he had severe neurological symptoms. The first E.E.G. showed a marked depression of all electrical activity—practically no cerebral activity at all. Slow waves appeared 2 months after the accident as in fig. 2. They increased in

voltage in the next $3\frac{1}{2}$ years. Seizure discharges were observed about 2 years after the accident. They started with spikes, and shortly afterwards bilateral spikes and waves appeared. In the following 2 years more and more epileptic activity was recorded (fig. 3). The patient had clinical seizures during this period. Then the seizures ceased and the seizure pattern in the E.E.G. disappeared, while the slow waves became faster and decreased in voltage. By $4\frac{1}{2}$ years after the accident the frequency was 7/sec. and the amplitude 40 microvolt, which means an almost normal E.E.G. (fig. 4).

The patient then had a gross degree of atrophy with enormous enlargement of the ventricular system (fig. 5).

The explanation in this case, and probably also in many cases of cerebral palsy may be as follows. At first these patients have a primary cerebral damage which if severe, as in the above case, gives rise to marked electroencephalographic disturbances, because if it is slight the E.E.G. may at first be normal. This is because the pathologically changed nerve cells in the first period sometimes do not give rise to any measurable pathological electrical activity. After a

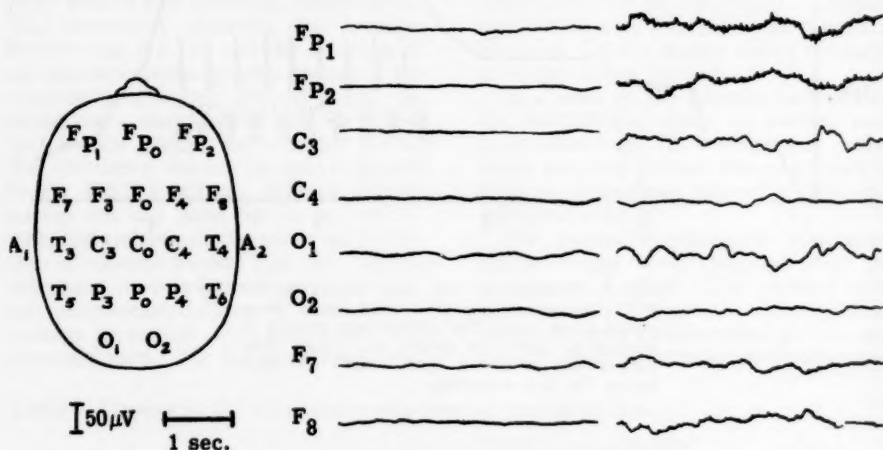


Fig. 2. The first "flat" EEG was taken 14 days after the accident and the second with slow waves 24 months after it. In all recordings the electrodes are placed as in this figure and the time marker shows seconds. The average techniques are used in all records.

while, however, these nerve cells will increase their electrical activity, as judged by the E.E.G. records, and the E.E.G. will

show slow waves. Some will even be hyperactive and become epileptogenic, and we may record sharp wave, or spike and wave.

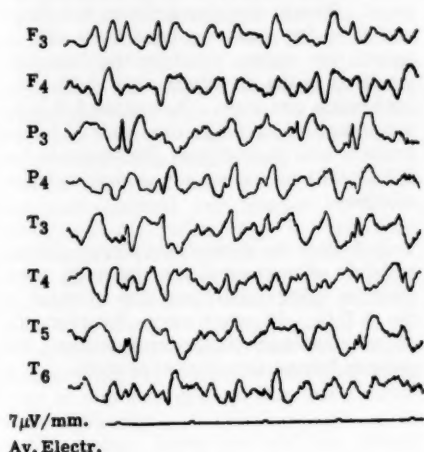


Fig. 3. EEG recorded 29 months after the accident. Sharp waves or spikes were then registered for the first time.

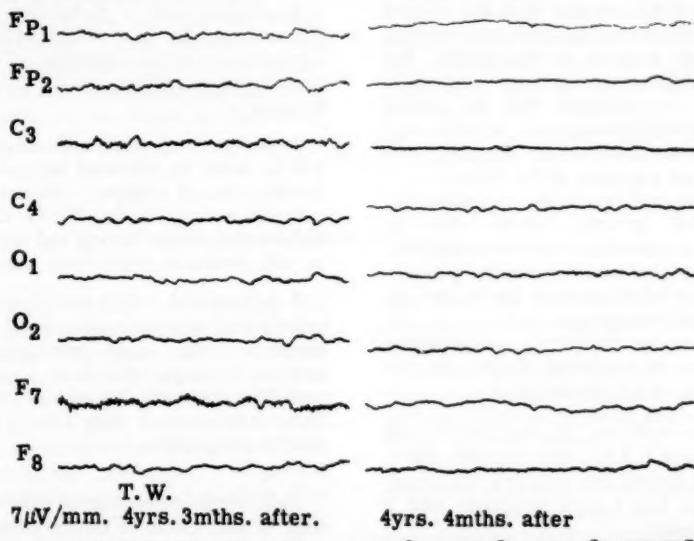


Fig. 4. The first EEG on the left was recorded 4 years 3 months after the accident and the second 4 years and 4 months after it, when the EEG looks almost normal.



Fig. 5. Post-mortem specimen showing gross atrophy with enormous enlargement of the ventricular system.

In this way the lesion will be progressive and the patient may or may not develop clinical seizures in this period. The pneumoencephalograms are sometimes normal in the first stages of the disease but are often pathological later on. In some patients the pathological nerve cells will after a while be replaced by glial tissue. The abnormal electrical activity will then disappear, since the glial cells do not produce any electrical activity in the E.E.G. The normal nerve cells, which may be markedly reduced in number, will then show normal activity, and the E.E.G. will seem normal. The pneumoencephalogram, however, will at this stage be pathological, and the neurological findings will be the same as before. The primary lesion has now become stationary and it will probably remain stationary for the rest of the patient's life.

It is therefore highly important to correlate the clinical, pneumoencephalographic and electroencephalographic findings in repeated examinations over a period of many years. We are now doing this in our clinic, and in a few cases we have been able to record the same electroencephalographic results as in the case mentioned above, thus confirming our view. By repeated E.E.G. recordings we have also been able to follow patients who show seizure discharges in the E.E.G. without having any clinical epileptic seizures.

In 5 cases the patients developed clinical epileptic seizures some months to 2½ years after the spike activity was first recorded in the E.E.G. In other cases, however, the seizure discharge disappeared without the patients having any clinical seizures.

CONCLUSION AND SUMMARY

A single E.E.G. record is in our opinion of little value in most patients with cerebral palsy, except in cases of hemiparesis. But repeated E.E.G. examinations, especially when they are compared with the clinical and pneumoencephalographic results, may give valuable information regarding the prognosis and the state of the lesion.

In this way we can follow the pathological cerebral process, which may be stationary, progressive or even epileptogenic. We can thus also record when the pathological activity of the process ceases and the lesion becomes stationary.

By correlating the E.E.G. pneumoencephalographic and clinical results we can draw the following conclusions:

A patient with one, or even better with several normal E.E.G.s and a normal pneumoencephalogram, and with few neurological systems, has a good prognosis and a stationary lesion.

A patient with one, or even better with several normal E.E.G.s but with a pathological pneumoencephalogram and with few or many neurological symptoms probably has a stationary lesion.

A patient with a pathological E.E.G., characterized by slow waves, probably has a progressive lesion, regardless of the pneumoencephalographic and neurological findings.

A patient with seizure discharges in his E.E.G. must be followed because he may develop clinical epilepsy. However, if the epileptic activity in the E.E.G. disappears without the patient having had any epilepsy he will probably never have seizures.

A patient with a local severe reduction in voltage and with no slow waves, but with marked clinical and pneumoencephalographic findings, will have a large and probably stationary lesion. If the E.E.G. shows slow waves or spike activity the lesion may be progressive.

Such triple measurement will reveal the state of the lesion. If this is progressive it may explain some of the increasing neurological and psychiatric symptoms and in some cases will account for the poor results of treatment as well as being of prognostic value regarding epilepsy.

RESUME

Un tracé d'E.E.G. isolé est à notre avis de peu de valeur pour la plupart des malades atteints d'infirmité motrice cérébrale, sauf en cas d'hémiplégie. Mais des examens répétés par E.E.G., surtout s'ils sont rapprochés des résultats cliniques et pneumoencéphalographiques, peuvent donner des renseignements de valeur, notamment sur le pronostic et l'état de la lésion.

C'est ainsi que nous pouvons suivre le processus cérébral pathologique et déterminer, par nos recherches, s'il s'agit d'un état stationnaire, progressif ou épileptogène. Nous pouvons aussi bien noter à quel moment le processus cesse d'être pathologique et la lésion devient stationnaire.

La corrélation des résultats électroencéphalographiques, pneumoencéphalographiques et cliniques nous amène aux conclusions suivantes:

Un malade ayant un seul ou, mieux encore, plusieurs E.E.G. normaux, et dont le pneumoencéphalogramme est normal et les symptômes neurologiques rares, a un bon pronostic et sa lésion est stationnaire.

Un malade ayant un seul ou, mieux encore, plusieurs E.E.G. normaux, mais dont le pneumoencéphalogramme est pathologique et les symptômes neurologiques rares ou nombreux, a une lésion probablement

stationnaire.

Un malade ayant un E.E.G. pathologique caractérisé par des ondes lentes aura probablement une lésion évolutive, quelles que soient les constatations pneumoencéphalographiques et neurologiques.

Un malade ayant des décharges paroxystiques à l'E.E.G. doit être suivi comme susceptible de développer une épilepsie clinique. Cependant, si l'activité épileptique disparaissait de l'E.E.G. sans que le malade ait eu d'épilepsie, celui-ci n'aura probablement jamais de crises.

Un malade dont l'E.E.G. présente une réduction locale notable du voltage, sans ondes lentes, mais chez qui les constatations cliniques et pneumoencéphalographiques auront été nettement marquées, aura une lésion importante mais probablement stationnaire. Si l'E.E.G. montre des ondes lentes et des pointes, la lésion peut être évolutive.

Ce triple procédé d'évaluation établira l'état de la lésion. Si celle-ci est évolutive, il peut expliquer certaines poussées neurologiques et psychiatriques et, dans quelques cas, expliquer le résultat modeste du traitement aussi bien que présenter un intérêt pronostique dans l'épilepsie.

ZUSAMMENFASSUNG

Eine einzige E.E.G. Aufzeichnung hat unserer Meinung nach geringen Wert bei den meisten Patienten mit zerebraler Kinderlähmung, ausgenommen in den Fällen von Hemiparesis. Wiederholte E.E.G. Untersuchungen aber, besonders wenn man sie mit den klinischen und pneumoencephalographischen Befunden vergleicht, können wertvolle Auskunft, vor allem über die Prognose und den Zustand der Schädigung geben.

Auf diese Weise können wir dem pathologischen Gehirnprozesse folgen, und erforschen, ob es sich um einen stillstehenden, fortschreitenden oder sogar epileptogenen Zustand handelt. Wir können auch festset-

zen, wann die pathologische Tätigkeit des Prozesses aufhört und die Schädigung zum Stillstehen kommt.

Aus der Verbindung der klinischen, elektroencephalographischen und pneumoencephalographischen Befunde können wir folgende Schlüsse ziehen:

Ein Patient mit einem, oder besser mehreren, normalen E.E.G. und mit normalen Pneumoencephalogramm und wenigen neurologischen Symptomen hat eine gute Prognose und seine Schädigung ist stationär.

Ein Patient mit einem, oder besser mehreren, normalen E.E.G. aber mit pathologischem Pneumoencephalogramm und wenigen oder vielen neurologischen Sympto-

men, hat wahrscheinlich eine stationäre Schädigung.

Ein Patient mit inem, durch langsame Wellen gekennzeichneten pathologischen E.E.G., muss, abgesehen vom pneumoencephalographischen und neurologischen Befund, wahrscheinlich eine fortschreitende Schädigung haben.

Ein Patient, dessen E.E.G. paroxystische Ausladungen aufweist, muss überwacht werden, weil sich bei ihm eine klinische Epilepsie entwickeln kann. Wenn jedoch die epileptische Tätigkeit aus dem E.E.G. verschwände, ohne dass der Kranke Epilepsie gehabt hätte, wird er wahrscheinlich niemals an Anfällen leiden.

Ein Patient, mit beträchtlicher örtlicher

Herabsetzung der Spannung ohne langsame Wellen, aber mit bedeutenden klinischen und pneumoencephalographischen Befunden, muss eine grosse, aber wahrscheinlich stationäre Schädigung haben. Wenn das E.E.G. langsame Wellen oder Spitzen aufweist, kann es sich um eine fortschreitende Schädigung handeln.

Dieses dreifache Abschätzungsverfahren wird den Zustand der Schädigung feststellen. Ist diese fortschreitend, so kann es sowohl manche neurologische und psychiatrische Verschlimmerungen und, in gewissen Fällen, die geringen Ergebnisse der Behandlung erklären, als sich auch von prognostischem Wert bei Epilepsie erweisen.

"EPILEPSIA"

Epilepsia has had a difficult time in the past 10 years, and when it was appearing only once a year its editorial committee thought it was serving no useful purpose. It is greatly to the credit of the Dutch publishing firm, Elsevier, that they have made it possible for this fourth series of the journal to appear quarterly and with much improved print, paper and illustrations.

Epilepsy is no narrow specialism. The research into its causation is of the greatest importance to the understanding of normal and subnormal brain function. The number of epileptics makes its treatment the responsibility of every family doctor, and the

reactions of the community to the epileptic affect us all. The editors, from France, U.S.A. and Holland, with Sir Francis Walshe as editor-in-chief, ensure the necessary breadth of approach, and the first number is most promising. It was odd not to find any reference to the changing social attitude to epilepsy, which seems so significant, but no doubt this will come.

*Epilepsia** in its new form should find a warm welcome.

DENIS PIRRIE

*Journal of the International League against Epilepsy. Fourth Series. Vol. 1. Annual subscription £2 17s Quarterly: 480 pp.

A CEREBRAL PALSY CENTRE IN THE TROPICS

C. ELAINE FIELD, M.D., M.R.C.P.

From the Paediatric Unit, the General Hospital, Singapore.

Dr. Elaine Field sets a clinical problem and gets some authoritative replies.

TO start from nothing, knowing nothing, is, I am sure, the best way to start a cerebral palsy centre. In Singapore, however, there was a beacon of light which set the fire burning and has since kept our enthusiasm alight through difficult times.

On October 10, 1956, Miss Paulette Leaning, a cerebral palsy teacher from New Zealand and herself a case of cerebral palsy, spoke to the Rotary Club in Singapore. She gave such an inspiring address and impressed her audience to such an extent with the mastery of her own handicap that afterwards members of the medical profession and Rotary Club met to discuss the possibility of establishing an Association to care for the cerebral palsy children in Singapore. The blind and the deaf had already received attention. There was, however, no-one with any special experience of cerebral palsy work, so the question was: How to start? To raise money and employ experienced workers from overseas was not really the answer; it was necessary for us to learn ourselves and adapt the most suitable methods for Singapore children. Through the Singapore Medical Department and under the auspices of the Colombo Plan, an experienced physiotherapist from New Zealand, Miss M. D. Hartridge, visited us for six weeks in February, 1958. Her information and advice were of great value and

started us on the right road.

Before her arrival, the Spastic Children's Association of Singapore, a voluntary organisation, was established on December 4, 1957. Composed of medical personnel, members of the Rotary Club and the interested public, the Association first established a class for the affected children. Readers may wonder why the term "spastic" has been used here in preference to "cerebral palsy". This is because "spastic" is more readily understood by the public, and their co-operation is essential.

Teething Problems

In Singapore there are three main races—Chinese, Indian and Malay. Over 80 per cent are Chinese, and this creates a language problem. Chinese and Tamil are difficult languages to learn, so Malay, now accepted as the national language, would be the language of choice. But, so far, we have been largely dependent on voluntary assistance, and many of these have been English speaking only—our problem deepens!

An early requirement was to improve the general health and nutrition of the children and gain their confidence. Many come from very poor homes and are just left to lie or sit at home. Then often there is no-one to bring them up to the classes because both

the mother and the father have to work. Our most urgent need, therefore, was for transport, so it was with considerable gratitude we accepted the gift of an ambulance from the Reuben Meyer Trust. Next came our biggest problem of accommodation for our classes. At first they were transferred from room to room in the General Hospital, until in desperation we rented a classroom from the nearby Y.W.C.A. centre. All the time we were trying various avenues to obtain a compound house at low rental. As I write, I am pleased to report our request has been met by the Singapore Government and we shall soon be moving into a large house. In addition, a grant has just been made by the Rotary Club of up to \$17,000 (Malayan) to equip our new quarters. The Centre, at last, is taking shape and we are most grateful to our benefactors.

Medical Problems

The scarcity of trained staff and little money to pay them creates another difficulty, which is being overcome in a remarkable way. All the patients are referred to the General Hospital, where they are medically examined by a paediatrician and their mental state roughly assessed. An almoner keeps a register of all these cases. The more intelligent ones are then assessed by a panel of voluntary qualified helpers, which include a physiotherapist, a speech therapist, a psychologist and, if indicated, an orthopaedic surgeon. An almoner visits the child's home to assess the social background. Our only paid worker is a qualified occupational therapist, who is supervisor of the classes. Once a month, the Case Committee, composed of these qualified personnel, meets to discuss in detail each new case, and to decide the best approach and care of that child. Children entering the special classes are first assessed by the Case Committee and then given six months' trial in the classes.

The causes of cerebral palsy in Singapore are much the same as elsewhere, but two predisposing conditions are high on the list—kernicterus and encephalitis. The causal factor for the kernicterus is not yet clearly defined, for rhesus incompatibility is rare. Prevention is possible by exchange transfusion, but many cases come too late for treatment. The causal factor for

encephalitis is variable; about 50 per cent are due to the Japanese B encephalitis virus. Unfortunately, many of these children are mentally subnormal. The incidence of cerebral palsy in Singapore, with a population of 1½ million, has been roughly assessed at 750 to 1,000. So far, on our register, we have 239 cases without looking for them. Most of these are under 12 years of age. An analysis of 100 of these showed 25 attending classes, 30 on the waiting-list, 33 attending a follow-up clinic, 8 mentally defective and 4 transferred to normal school. This is, however, a somewhat selective group because we have chosen the more intelligent for review first. The mentally defective group is, I should say, as large as in other countries.

The Classes

Starting with a group of 7 children in 1957, our number has now increased to 25, with ages ranging from 3½ to 11 years. There are three classes — primary A, primary B and kindergarten — and these have had to run in sessions because we have only one classroom. The supervisor of classes is a trained occupational therapist and is paid, but all the other helpers give their services voluntarily, some of them being trained teachers. The coordinator of voluntary helpers arranges a roster, so that the classes are always well staffed. This voluntary assistance has been invaluable.

The Future

A five-year-plan was drawn up a few months ago. In this plan it was proposed to expand the special classes to accommodate 50 children and later to start a small residential unit for about 10 children, particularly for those problem cases which require observation away from their home surroundings. Within the five years, it is hoped to start an occupational workshop for up to 10 adolescents, since we feel that fitting the children for a suitable job is especially important. It is hoped to extend outpatient follow-up clinics for those not attending the classes and to include parent groups at the new centre, and also to improve the treatment facilities for all, particularly physiotherapy, occupational therapy and speech therapy. This is a big programme and an

expensive one, particularly when we are entirely dependent on voluntary appeals for our funds, but the effort is so worth while. To see their faces as they change from timorous, frightened children to happy con-

fident ones after a few months in the classes is a joy in itself. We are confident, with the assistance of our good friends here in Singapore and overseas, that we shall accomplish our aims and complete our five-year-plan.

A Problem

One child, aged 5 years, a severe athetoid, has set us a problem which I am sure your readers would like to answer. Mentally normal, she is unable to use her hands because of producing severe spasms, but she can use her feet surprisingly well by feel as she cannot at the same time turn her head

to look down because of a persistent tonic neck reflex. Do we encourage the use of her feet, thus making her a bit of an oddity, or do we try to overcome the spasm created by using the arms and discourage the use of the feet at present?

. . . and some Replies

From Dr. N. S. Alcock

The crux of this problem is the severity of the arm spasms. If at the age of 5 they are so severe as to prevent any useful activity then the prospect of modifying them by any medical means is extremely remote. There is a possibility that thalamectomy or pallidectomy might help but so far results in athetosis have been disappointing.

It is only too easy to discourage foot skills and very difficult to re-establish them once lost. Despite their low social acceptance I would feel that they are probably the only way in which the child will do anything for herself and I would be very reluctant to abandon them.

In some cases we have found that the use of the feet will exacerbate the tonic neck reflexes and by so doing may prejudice the development of normal speech. If in this child this happens then foot skills are not to be valued above speech.

From Dr. Marcel d'Avignon, Stockholm

You say that this child with severe athetoid cerebral palsy is mentally normal but is unable to use her hands, because of producing severe spasms, though she can use her feet surprisingly well. I think it will be very difficult to overcome the spasm created by using the arms, at least for the next few years. I hope when the child gets older that the athetoid movements and the changes

in tone, from hypotonus to rigidity, will be less pronounced. If her feet are in a relatively good state I think these should be trained.

We know such patients who can do quite a lot with their feet, such as painting. Perhaps it is impossible now to train the upper extremities, especially the hands. When a child grows older and has been under cerebral palsy treatment for a certain period it is very often easier for them to relax, and at the same time when the child gets older perhaps the symptoms will spontaneously regress a little, and treatment of the hands will be possible. I think that all possibilities to treat such severely handicapped children should be exercised. Have any auditory tests been performed?

From Mrs. D. P. Beaman, C.S.P.

Dr. Elaine Field writes here of one of the greatest problems in the field of cerebral palsy—a useless pair of hands! We all know that it is only through their use that we can achieve a full independence, even against the background of a wheel-chair existence.

In substituting feet for hands, the question of “making her a bit of an oddity” should not colour the therapist's decision. With the described degree of handicap, her appearance will be far from normal anyway. Of greater importance, therefore, is the fact that she should be capable of expression in life.

Usually with this type of lesion the speech is intelligible, though with effort, and if the intelligence is normal she should have a potential ability to have full contact with people. This, therefore, is her first threshold, her most treasured ability, and of greatest importance. In using feet for intrinsic control, the activity of the T.N.R. would surely increase, and though the effect upon the hands would be of no importance, it could certainly complicate progressive development of speech control.

Inadequate use of feet with excessive effort and poor results, may well produce further frustration with age, endangering her sense of well-being, whereas if she could be helped to accept full physical dependence, lessening the physical effort while channelling activity from an early age into intellectual perception and expression, one feels that life would be more satisfying for her.

On the other hand, had her I.Q. been lower, the resultant unambitious passivity may well have been satisfied with this sub-normal level of achievement.

One cannot generalise from a distance of 10,000 miles, and the longer one works in this field, the less dogmatic we become, as we search fruitlessly for two identical cases. But in similar cases to this, in normal I.Q.'s, we have disregarded potential foot control for the reasons given above, and through reducing the T.N.R. influence, have helped, I hope, towards a more important type of independence—the ability of intellectual expression.

From Mr. G. A. Pollock, F.R.C.S.

From your correspondent's note I gained the impression that this child corresponds to one of Phelps' 12 types of athetosis—that with "educated feet"—and can therefore be classified as a head and neck athetoid. This is a very characteristic group. I have seen three, and the term "educated feet" is an apt description undoubtedly.

My suggestion would be that full training to develop the activity of the feet should be persevered with, since some of these patients become most adept with a typewriter; others can paint, and some can even thread a needle—but their's is not the type of ability

that one would be inclined to boast of socially. Nevertheless, if an athetoid is capable of using his lower limbs in an economically gainful manner, or even for feeding, how much better is his lot than that of the average athetoid who can do very little!

So far as the upper arms are concerned, I do not think any improvement will occur until the tonic neck reflex is abolished, or at least diminished, and exercises towards that end should be continued.

From Dr. Guy Tardieu, Paris

This little girl is probably an example of the form of cerebral palsy described by W. Phelps as "neck and arm athetosis". Two examinations are necessary: (1) for Rh factor as an aetiology; and (2) for partial deafness hindering the hearing of sharp sounds.

As a rule in such cases, the children are much more skilful with their feet than with their hands. I have seen some of them weave, write, paint or type with their feet. The involvement of the upper limbs varies from case to case. Some of the children are definitely unable to use their hands, and we have then to be content with the kind of activity mentioned above. Others can use their hands a little.

I have not seen the present child and cannot give a definite opinion. But I would say that in everyday life we should let her use her lower limbs as she likes, so as to give her a feeling of achievement. Since she is intelligent we may, though **only during the 30 minutes of occupational therapy**, ask her to make personal efforts to use her upper limbs. If no results are obtained after a while it would be bad to insist too much, if the child has an anxious feeling of unsuccess. Of course, general methods (classical, such as relaxation, or Bobath techniques) may also be used.

From Dr. Grace Woods

At the Bristol School for Spastics there was a similar problem two years ago in a girl of 7 years. She was a case of athetosis with marked spasm. Owing to exceptionally strong tonic neck reflexes, when undressed

her arms snapped forward in flexion, and there were scratch marks on her chest-wall from her own fingers which she could not control. She had, however, fairly good use of her feet, and would, for example, play with sand using her bare feet. Feeling hopeless about the future usefulness of her hands, the staff of the school had decided to allow her to use her feet. The case, however, was seen in conference with Dr. Marjorie Wilson of the Ministry of Education, and she strongly advised against making the child an oddity at this early age. This advice has been followed; and we feel the policy was justified. By methods of physiotherapy aimed at reducing the tonic neck reflexes and the strong spasm in the arms, this child can now

use a typewriter fairly usefully for school work, and has also learnt to walk.

Another factor in this child's improvement has been the administration of 'Mysoline' (primidone). It has been found in Bristol that some athetoids with severe spasm have improved on small doses of this drug, of the order of 25mg. twice a day, or less. The response is variable, but it is worth trying in every case. The spasms in this child were more pronounced when the drug was discontinued for a month. In the present case Mysoline might be of assistance, but the grounds for persevering with treatment of the upper extremities seem even stronger than in our case, for this child cannot see her feet and this would reduce their usefulness.

Postscript

Dr. Field to The Editor:

I found the replies to my question most interesting and helpful. It is still difficult to know the best line of treatment, but, after discussing the problem, with these replies, in our Case Committee meeting, we have decided to continue to encourage the child to use her feet, but at the same time we shall intensify the physiotherapy on her arms and neck to try to overcome the tonic neck reflex. Once this is overcome, I feel we should encourage her to use her hands just as much as, if not more than, her feet.

She is improving quite considerably since attending our special classes, and we have every hope that she will make good progress.

THE NEW MENTAL HEALTH ACT

ROBINA S. ADDIS.

*From the Social Services Department, National Association
for Mental Health, London.*

The Act provides a charter for the care, treatment and training of the mentally disordered, and everyone working with the mentally subnormal should understand its provisions. This general review is intended to help them to do so.

The Mental Health Act 1959 scraps all the previous legislation connected with mental deficiency and indeed abandons the name. New terms are substituted, new definitions made and new machinery set up. For all those interested in work with the mentally subnormal, it is necessary to understand the working of the Act, sections of which are now beginning to come into force and here an attempt will be made to outline its main provisions for the consideration of workers in this field.

First, a single code is applied to all cases of mental handicap and disturbance, and these are dealt with by the one Act, instead of, as previously, by separate legislation. Under the term "mental disorder" are included cases of mental illness, mental subnormality and a new category of psychopathic disorder. It is curious that the man in the street has obstinately clung to his description of "mental" for any person who differed from the normal, whether by lack of intelligence or strange behaviour, and our attempts to persuade him to distinguish between mental defect and illness have been in vain. Now he can apply "mental disorder" indiscriminately and be correct.

In this Act "mental disorder" means serious mental illness, arrested or incomplete development of mind (divided into "severe subnormality" and "subnormality"), psychopathic disorder, and any other disorder

or disability of mind.

The Act makes a heroic attempt to meet some of the difficulties which arose from the old categories of mental deficiency. Those severely handicapped, the idiots and imbeciles, and some of the feeble minded have been generally acknowledged to need special care and treatment and to be unable to lead independent lives, but the higher grades might be able to fend for themselves yet could not conform with social or moral codes, and it has been difficult to decide at what point restriction becomes necessary.

The old "moral defective" category in the 1927 Act was defined as follows:

Moral defectives, that is to say, persons in whose case there exists mental defectiveness coupled with strongly vicious or criminal propensities and who require care, supervision and control for the protection of others.

This was generally taken to include persons with normal or even superior intelligence, who would now be called psychopaths. There was no way of dealing with them under this category except to admit them to mental deficiency hospitals where it was difficult to provide any special treatment for them and where they associated with mental defectives which might be to their detriment. It was this section of the population of these hospitals which led to the surprisingly high average intelligence in these institutions for the subnormal.

The "psychopathic personality" category will now cover the mentally abnormal of anti-social tendencies who do not respond to ordinary methods of treatment and training, whether they are or are not also of sub-normal intelligence. With the better definition of the problem and the grouping of cases, it is hoped that improved methods of dealing with those intractable conditions will be devised. The special provision for treatment of the younger cases suggests that the period before the age of 21 years is thought to be the more favourable. Here lies opportunity for experiment and variety of approach. It is to be hoped that group therapy, which has already given promising results with some anti-social cases, as well as individual treatment, will be explored and that various forms of therapeutic environment will be provided so that different approaches can be tried out.

Designated Hospitals

The second main change introduced by the Act is the abolition of "designated hospitals". Previously, mentally ill and mentally defective patients could be admitted only to certain hospitals set aside for the purpose. Moreover, these hospitals were bound to admit any patient provided he was submitted on the properly completed legal form. In 1930 the Mental Treatment Act allowed voluntary patients to enter mental hospitals, as well as those on certificate, but they also had to sign a legal form and could not be accommodated except in a place approved for this type of patient.

All this is now altered and there are no longer to be any designated hospitals, any kind of hospital can admit any kind of patient, informally or under detention. Revolutionary though this may appear, there was already a trend in this direction as can be seen from the establishment of psychiatric units in certain teaching and general hospitals. It was a logical sequel to the opening of psychiatric out-patient clinics in general hospitals, that the psychiatrists in charge should look for beds directly under their care. Thus it was proved that many cases of mental illness or neurosis could very well be treated within a general hospital. There need not be anxiety about the

possible effect on the other patients, because under the new Act it is open to the hospital to accept or reject the admission of the patient, and responsible doctors must be relied on to safeguard the interests of all the patients.

Although the Act has not yet come into force, informal admissions of mentally disordered persons are already permissible alongside the old machinery for admissions. Since January 1958, mental deficiency hospitals have admitted patients without formality, and this procedure was applied to mental hospitals in October 1959. Here we have an attempt to approximate measures for treatment of the mentally disordered, with those for cases of physical illness or injury and this was recommended in the Report of the Royal Commission. It has great advantage in making it easy for patients to accept hospital treatment without unnecessary fears and we know that for many psychiatric patients these formalities proved a stumbling block. Naturally, there is a dread of being "shut up" or "put away" and for the subnormal or the mentally disturbed, a legal form may seem a threat.

Informal admissions mean informal discharges and some popular concern has been expressed lest patients who need to be under care or treatment may walk out of hospital to their own detriment or to the disturbance of others. If we are to encourage easy access to treatment, this responsibility must be accepted. After all, if the present voluntary patient takes his own discharge without waiting for the 72 hour notice which he undertook to give, he is allowed to go unless it is necessary to issue a "barring certificate". This medical responsibility still exists and if the patient is a potential danger to himself or others, steps can be taken to detain him.

Compulsory Admission

This brings us to the question of compulsory treatment. In spite of attempts to approximate the care of the mentally disordered to that of the physically sick, and we have seen the advantages of this approach, there are particular aspects of psychiatric cases which require special measures. It is acknowledged that there are

certain types of case which are either incapable of expressing acceptance of treatment or are hostile to it, and where compulsory detention is necessary, the Act makes detailed provision for this. The necessity for legal documents requiring a magistrate's signature is abolished and a solely medical responsibility established.

A patient suffering from mental disorder and who "ought to be so detained in the interests of his own health or safety, or with a view to the protection of other persons" can be detained for observation for a period of up to 28 days on the recommendation of two doctors. In an emergency he can be admitted for up to 72 hours on the recommendation of one doctor, on the application of a mental welfare officer or any near relative.

For compulsory admission for treatment the nearest relative or Mental Welfare Officer makes application and two doctors who have recently examined the patient complete the form. One of these doctors must be approved by the local health authority as having special experience in the diagnosis or treatment of mental disorder, and unless he has "previous acquaintance" with the patient, the other medical practitioner shall if practicable already know the patient. Thus an attempt is made to have medical recommendations, based both on psychiatric skill and knowledge of the individual patient. Both doctors have to state their grounds for their opinion on the patient's condition and specify why other means of treatment are not available.

These safeguards are for the protection of persons who may be considered to require detention during treatment. An application for admission for treatment may be made in respect of a patient of any age suffering from mental disorder which is diagnosed as mental illness or severe subnormality. If he is suffering from mental disorder and is under the age of 21, patients diagnosed as cases of psychopathic disorder or subnormality may be admitted for compulsory treatment. This is the provision for the early treatment and training already mentioned as applicable to the old category of "moral defective" and will include the disturbed and anti-social feeble-minded. The Courts also have power to send a person of any age convicted of an offence and found to be suffering from mental disorder, to a

hospital for compulsory treatment.

Guardianship

As an alternative to detention for treatment in hospital, the Act provides for guardianship under the local health authority or other person accepted by them. Here the mentally disordered person will be living in the community without restriction except in so far as it can be imposed by the authority of the guardian. Regulations will be issued at a later date, but the Act itself throws little light as to how this will work out. It states that the guardian will have such powers "as if he were the father and the patient under 14 years old", and there is a reference to "the place at which he is required by the guardian to reside". In dealing with a difficult psychopath, disturbed adult or wayward mental defective, such requirements may be difficult indeed to enforce. Supervision in the interests of the patient will also be necessary, and the Act provides for the mental welfare officer to be able to apply to the County Court if he considers the guardian is negligent or is acting contrary to the interests of the patient.

Guardianship has not previously been applied in this country to cases of mental illness but only to mental deficiency, and although the M.H. Act makes provision for its use, previous figures have shown a marked decrease. The latest Report of the Minister of Health gives the number of mental defectives under guardianship or notified as 2,181 for 1958 compared with 2,939 for 1957. It is strange that this declining provision as applied to the 142,619 defectives under some form of care, should be extended by the new Act to cover a much wider field. Adequate social services, hostel accommodation and the support of the community will all be required to make guardianship a reality.

Provisions for Review

Applications for compulsory treatment under hospital care or guardianship are limited to fixed periods and expire on the end of the first year unless renewed for a further year. After that they last for two-year periods. This should ensure that there are no "forgotten" patients unnecessarily detained. Those who fear that disturbed

patients may be too readily released may be reassured by the clause which provides for each case to be reviewed two months before the expiration of the order. It is the duty of the responsible Medical Officer to report to the hospital managers (or to the local health authority in guardianship cases) if "in the interests of the patient's health or safety or for the protection of other persons that he should continue to be liable to be detained". This also applies to the patients detained as psychopaths or subnormal who have to be discharged at the age of 25 years, unless likely to be a danger to others or themselves. We must rely on the high standards of medical responsibility to allay the fears of those who see a danger in this age limit.

The public has expressed fears about the effect on the community of prematurely discharged patients, in other contexts it has also questioned the preservation of the liberty of the subject if investigation by a magistrate is no longer required before a patient's freedom is removed. In leaving the decision as to compulsory treatment entirely in medical hands, the Act still allows for the patient's case to be considered by lay persons. Mental Health Review Tribunals are to be set up to hear appeals from patients and their relatives and will have power to release. They are to be composed of legal and medical members plus others of "such experience in administration, such knowledge of social services or such other qualification or experience as the Lord Chancellor thinks suitable".

These Tribunals are a new conception and allow of the personal appearance of the patient and his relatives to voice any grievances about detention. Since it is characteristic of many psychiatric patients to think themselves wrongfully detained, the Tribunals may expect to be faced with more problems of mental disorder than of civil liberties, but it would seem a wise protection for the rights of patients, particularly since the Act also does away with the old Board of Control which had many watch dog functions. Membership of these Tribunals will offer opportunity for public service and should be taken up by those experienced in the problems of mental disorder, and especially in the social problems.

The Court of Protection is preserved for

the protection and management of the affairs of persons "under disability" and will continue to look after the financial interests of patients unable to manage for themselves. Restrictions may be made as little onerous as possible, but there is one which has been retained and has caused some comment. Censorship of letters is allowed in certain circumstances and some critics have been anxious lest this should lend itself to abuse. The managers of a hospital, or the guardian may return a packet to the sender if it would cause the patient distress, or withhold a patient's letters if it would be "unreasonably offensive" to the addressee, defamatory to other persons (with the curious exception "other than persons on the staff of the hospital") or likely to prejudice the interests of the patient. Letters to the Minister, members of Parliament, the Court of Protection, the managers of the hospital and the Mental Health Tribunal (at any time when the patient is entitled to make application to it) are privileged and must be posted.

Interests of the Public and the Community

Throughout the Act can be seen an attempt to balance the interests of the patient in his need for treatment and his claims for liberty, as well as to safeguard the community. Much of the Act is necessarily taken up with the legal setting out of conditions for compulsory admission to hospital, but its effect is to allow as much informal treatment as possible and to place the emphasis on community care. Part II deals with the Local Authority services and begins by stating that Section 28 of the National Health Service Act 1946 (which relates to functions of local health authorities with respect to the prevention of illness and the care and after care of patients) shall have effect in relation to persons who are, or have been suffering from mental disorder. The Minister has told us that this hitherto permissive power is to be made obligatory, and local health authorities are required to submit schemes by April 1st, 1960 stating how they propose to carry out the various duties consigned to them. The Mental Health Act enlarges on the local authority functions in stating that they include:—

- (a) the provision of residential accommodation.

- (b) the provision of training or occupation centres.
- (c) the appointment of Mental Welfare Officers.
- (d) the exercise of guardianship functions.
- (e) the provision of ancillary or supplementary services.

These wide powers should allow of any and all services required for the care in the community of mentally disordered patients. Satisfactory social services will be the key to success of such schemes and it is to be hoped that following the recommendations of the Younghusband Report, the Mental Welfare Officer will be a qualified Social Worker and equipped to give an adequate case-work service.

Care of Children

A welcome change brought about by the Act is the removal of barriers from the care of mentally disordered children who may also be deprived of family life. The Children's Department is now permitted to receive into care any child who is mentally disordered, or if such a child or young person is already in care and admitted to a hospital or nursing home, the local authority is required to arrange for visits, "or such steps as would be taken by parents". In addition to this, the Children's Authority may accommodate in homes or elsewhere any child who is not in care but is being dealt with under Section 28 of the N.H.S. Act (see above), which relates to prevention, care and aftercare.

Some apprehension has been expressed lest Homes for deprived children will become burdened with cases of mentally disordered children for whom they have neither staff nor facilities. No responsible department would place a handicapped child in these conditions and we must rely on satisfactory representation between staff and the administrative body, as is necessary for all admissions. Moreover, it is to be hoped that adequate training and treatment facilities will be provided so that these cases will be sent to the appropriate place. The advantages in permitting the local authority to act

in loco parentis for every child deprived of family life, whether it is mentally handicapped or not, greatly outweigh the possible dangers. Those responsible for the care of deprived children and those whose concern is for the mentally disordered child, should be constantly on the watch to see that the best is done for every case.

One other function of the local authority in this field is the provision of training centres for the mentally disordered children found unsuitable for education at school. The Act now gives them power to compel the attendance of school-age children either by day or as a resident. The authority before giving notice under this section must be satisfied that the child is not receiving comparable training elsewhere, but even with this possible escape, parents who would be adverse to letting their child go away from home may feel anxious. Unless more training centres are provided so that every child could attend by day, this possibility of compulsory removal from home may be a threat. It can only be said that in a comparable situation where a child is recommended to attend a boarding school for maladjusted pupils and the parents resist, the law is never invoked.

The Community Part

In ending on the local authority functions within the new Act, this paper underlines the emphasis on community care. If the hospitals discharge these patients who need only residential support, they can devote themselves to the highly specialised medical treatment which is required for some patients. The community must then take up its responsibilities and here most opportunity arises for the active help of people of good will. The Act provides a Charter for care, treatment and training of the mentally disordered. Its enlightened provisions will be of no avail unless they are carried out by personal service in a sympathetic environment. Not only the hospital services and the local authorities have duties to perform. Responsibility rests on the community to share in making the necessary provisions and to give individual help to the mentally disordered.

RESEARCH BACKED BY THE NATIONAL SPASTICS SOCIETY

ALISON D. MACDONALD, M.D.Lond., D.P.H., D.C.H.

From the Department of Child Health, Guy's Hospital, London.

A brief survey of the work done since 1953 by research workers receiving grants from the Society, as recommended by its Medical Advisory Committee, as well as by the Society's Medical Research Unit at Guy's Hospital; with some account of other activities fostered by the Society as likely to advance and spread knowledge over a wide field centred on but by no means confined to cerebral palsy.

THE study and alleviation of cerebral palsy requires an approach through many disciplines. The name encloses a heterogeneous group of neurological disorders of children which is too wide for some purposes and too narrow for others. From the point of view of treatment and management cerebral palsy is a reasonably satisfactory and practical category of handicapped children, though education and training must depend on the individual child's physical and intellectual capacity.

From the point of view of causation the syndrome is too narrow, since it is but one of many congenital abnormalities which may be aetiologically related.

Research in cerebral palsy is directed towards two ends: to increase our knowledge of the causes and mechanisms, in order that preventive action may be taken; and to study cases of cerebral palsy with the object of perfecting methods of treatment and management which will enable cerebral palsied people to live as full and satisfying lives as possible.

Research into causes and mechanisms may be of different types. Epidemiological surveys, for example, can demonstrate associations between events at or before birth and cerebral palsy appearing later, and may form the basis for a hypothesis which can then be tested in another way. At the same time research effort needs to be directed towards a greater knowledge of the developing foetus, both physiological and anatomical, particularly its nervous system and ways in which environment and genetic background may affect development. Interactions between foetus and mother must especially be studied. An understanding of basic principles and physiological mechanisms should logically precede, or at least accompany, the study of the disease process. Thus research which may sometimes have no obvious connection with cerebral palsy is of fundamental importance to the understanding of this and other types of defect.

Since 1953 the Medical Advisory Committee of the National Spastics Society has considered and made recommenda

tions on applications for grants for research. In 1955 the Society appointed a Research Physician. As the volume of work increased further expert opinion was needed and in 1957 a Consultative Research Committee was formed. In the following year a Medical Research Unit was created, in association with the Department of Child Health at Guy's Hospital. The Research Physician became Director of the Unit and two Research Fellows were appointed.

Apart from the work of the Medical Research Unit, and the work supported by the grants, the Society has fostered various activities likely to advance and spread knowledge.

A sub-committee on Intellectual Assessment, comprising psychologists, paediatricians and psychiatrists, has drawn up a memorandum on the assessment of educational needs of children with cerebral palsy, and has made a valuable contribution to a pro-forma used for recording the history and clinical examination of cerebral palsied children.

A clinical classification of cerebral palsy is essential to any clinical or epidemiological research. The Little Club, composed of neurologists, paediatricians and other interested doctors, was asked to consider the definition and classification of cerebral palsy, and as no existing scheme seemed adequate it suggested guiding principles for a new system which might be useful to others working in this field. Their Memorandum on Terminology, published in *Cerebral Palsy Bulletin* No. 5, should assist research by enabling comparisons to be made between different series of patients.

An exchange of ideas on this and many other subjects associated with research in cerebral palsy was made possible by the holding of an International Study Group on Child Neurology and Cerebral Palsy at Oxford in September, 1958. This was a meeting of experts in various disciplines and from various countries and the discussion centred on neurological problems in children and infants. Much of

its proceedings have appeared—sometimes in reviewed form—in this *Bulletin*, and a general report on the meeting appeared in *Bulletin* No. 4 (Winter, 1958).

Liaison was established in 1957 between the National Spastics Society and the British Council for the Welfare of Spastics, and collaboration between the two societies is now becoming complete. Joint meetings have been held and the two Medical Advisory Committees have met at intervals. A joint plan has been drawn up for the neuro-pathological study of brains of cerebral palsied children. This is obviously desirable, since hitherto few brains, apart from those of mentally defective spastic children, have been examined in detail.

Studies undertaken by the Medical Research Unit

(1) *Survey of Cerebral Palsy in Britain*

Visits by the Research Physician to local health authorities in various parts of Britain have given valuable information on the number of children with cerebral palsy, methods of diagnosis and classification, and provisions available for treatment and education. Personal contacts and opportunities for discussing ideas for research have contributed towards evolving plans for research to which considerable time has been devoted since the inception of the Unit.

(2) *Perception in Normal and Cerebral Palsied Children.*

The education of many cerebral palsied children is apparently hindered by their inability to appreciate relationships in time, space and shape, and their awkwardness in mental manipulation and the handling of numbers. These disorders present a difficult problem, and much of the literature on the subject is confusing. As a first stage a research fellow in the Unit is making a study of reports relating to perception in cerebral palsied and normal children.

(3) *Studies of Developmental Mechanisms.*

(i) With others the Director of the Unit has shown that in certain apparent males who are sterile and females who fail to develop secondary sexual characteristics there are abnormalities of chromosomal constitution. By studying the incidence of colour-blindness, which is a sex-linked characteristic, these workers postulated the nature of the chromosomal aberrations. Recently the development of new techniques of study of human chromosomes has allowed them to confirm their suggestions. The findings are not only of general biological interest but are directly relevant to the field of nervous disorders, in view of the high incidence of intellectual subnormality among sufferers from these "sex-inversions". Furthermore, similar chromosomal anomalies have been shown to operate in mongolism.

(ii) The technique of "nuclear sexing" is at present being applied to cerebral palsied children, persons with mongolism and anencephalic stillborn babies. Some biological aspects of anencephaly are being studied with the help of workers in this country and at the Carnegie Institution of Washington, D.C. A statistical study of this condition has been planned in association with the National Birthday Trust Fund.

(iii) A comprehensive study of the normal chemical and enzymic development of the human nervous system, during both the early and late stages of pregnancy, is contemplated if a pilot study now being conducted proves successful. Preliminary biochemical and enzymic studies of embryological material supplied from Sweden are being made with the co-operation of the Department of Chemical Pathology, Guy's Hospital.

(iv) Embryological material is also being supplied from the same source to the Department of Neuropathology at the Maudsley Hospital, London, for morphological studies of developing muscle

spindles in human embryos.

(4) *Clinical Studies.*

(i) A simple electronic timing device for the measurement of reaction time and speed of movement has been constructed and tried out and is now ready for experimental use as an objective method of measuring motor handicap and assessing its possible modification by treatment.

(ii) A study group of experts in physical medicine is considering both the theoretical basis of major differences in technique and the possibility of a trial of the efficacy of different methods. A conference is contemplated if the preliminary work of the study group suggests that this would be fruitful.

(iii) The pro-forma designed for recording history and clinical examination of cases of cerebral palsy has been used in the Cerebral Palsy Advice Clinic at Guy's Hospital and elsewhere and is providing valuable information for research purposes.

(iv) Attention is being given to the neurological examination of newborn infants. Using methods described by André-Thomas in Paris, an attempt is being made further to develop the technique and recording of the examination.

(v) A study of palm prints of cerebral palsied children and where possible their sibs and parents is being undertaken. This study may aid the detection of abnormalities of intrauterine environment acting early in pregnancy and perhaps reveal evidence of genetic factors in some type of cerebral palsy.

(5) *Epidemiological Studies.*

(i) A large proportion—perhaps 50%—of cerebral palsied children are born prematurely, and the smaller the baby the greater the risk of cerebral palsy. In an attempt to find out what are the characteristics of the premature babies who develop cerebral palsy as compared with those who do not, over 1,000 children born in 1951-1953, weighing 4 lb.

or under at birth, and surviving beyond the age of six months are being followed up in conjunction with the Society of Medical Officers of Health.

(ii) Another method of studying birth factors epidemiologically is being used. A large series of known cases of cerebral palsy born in hospital is being collected with the help of parents, hospitals, and cerebral palsy clinics. Their birth records are examined and at the same time a control series of births at the same hospitals are collected. This should enable a statistical examination to be made of birth factors in large groups of clinically similar cases.

(iii) Toxaemia of pregnancy is often incriminated as a cause of cerebral palsy, but it is not known whether there is any higher incidence of cerebral palsy among children born to toxæmic mothers than would be expected from their degree of prematurity. A pilot study is being made at Guy's Hospital in co-operation with the London County Council and other Local Health Authorities in which children of mothers with severe toxæmia of pregnancy are compared with a control series of mothers who did not have toxæmia. If, as a result, a full-scale survey seems likely to give an answer to the question, the study will be extended to other hospitals and geographical areas.

(iv) About 3,000 children are being traced in a prospective study originally conducted by one of the research fellows in the Unit. The object of the investigation is to examine the relationship between maternal health in early pregnancy and congenital malformations and various neurological and other disorders in childhood.

(v) Twins provide a time-honoured means of distinguishing between the effects of nature and nurture. Furthermore, the incidence of twinning is apparently increased in cerebral palsy and there are other peculiarities in this condition that are related to twinning. A series of twins in which at least one of each pair has cerebral palsy is being examined with a view to establishing what is the incidence and type of cerebral palsy in the other children of each pair in identical and fraternal twins.

(vi) The Medical Research Unit is collaborating in a survey of cerebral palsy and neurological disorders in a national sample of children born in 1946. All children who show possible signs of cerebral palsy, epilepsy or mental defect are to receive a full clinical examination. This study will have particular value in describing the range of childhood neurological disorders in the community.

Bibliography on the Foetal Effects of Maternal Disorders

Miss Elizabeth Koenig, reference librarian of the U.S. National Institutes of Health, has compiled a selective bibliography of papers published in English between 1952 and April, 1959, on maternal disorders, with special emphasis on foetal stress, perinatal death and congenital defects. The N.I.H. is publishing the little booklet (33 pages, divided into eight subject sections, with an author index) "as a contribution to the common aim — increasing knowledge con-

cerning the effect of maternal disorders on the offspring and the prevention of needless loss". The bibliography will certainly help to focus attention on recent studies of this fascinating subject.

This is No. 25 of the Public Health Bibliography Series and is obtainable for 15 cents a copy from the Superintendent of Documents, U.S. Government Printing Office, Washington D.C., U.S.A.

REPORT

DEVELOPMENTAL DISORDERS OF THE CENTRAL NERVOUS SYSTEM

Research Reports given at the Annual Meeting of Czechoslovak Child Neurologists,
Marienbad, Czechoslovakia, June 11-12, 1959.

Reported by DR. IVAN LESNY (Prague).

We are glad to publish abstracts of the papers given at the annual Child Neurology Meeting in Czechoslovakia. Some of the papers may later appear in full in this Bulletin. Meanwhile this report of work in progress will stimulate by its example and will also enable workers in various fields of child neurology to exchange views with their Czechoslovak colleagues, either by letter or by personal visit.

Follow-up Studies of Children Born by Abnormal Delivery.

By I. LESNY and B. KULIS (Prague).

At a maternity clinic in Prague, 74 children (44 boys, 30 girls) born by abnormal delivery in one year, were examined at the age of 2 years; 23 of them were normal and 48 showed slight changes, mostly delayed development. Only 3 had a real cerebral palsy; in these cases there was not only abnormal delivery but another cause too, usually an infection in early infancy. These results suggest that abnormal delivery causes slight damage to the central nervous system which, when another lesion occurs, may result in cerebral palsy.

The Cause of Cerebral Palsy.

By J. KRAUS and E. OPATRYN (Zeleznice).

This is an etiological study of 768 children with cerebral palsy mostly boys (3:2). Clinically the most common type was hemiplegia (30 per cent), the right-sided ones predominating. The authors stress the frequency of prematurity (34 per cent) and of the first delivery (49 per cent). They conclude that combinations of factors are important in the causation of cerebral palsy.

Protracted Study of the Development of Children Suffering from Perinatal Trauma.

By G. BARDOSOVA and S. SRSEN (Kosice).

Continuous observations have been made of the psychomotor and neurological development of a group of children who showed symptoms of trauma during delivery and during their neonatal stay at the maternity home. The longest period of observation was up to the age of 5½ years.

The findings emphasise that neither the quality nor the severity of the symptoms exert so much influence on the child's development as their duration.

Though the numbers are small, the percentages show that the incidences of motor defects and delayed mental development were highest in the children whose postnatal symptoms lasted up to the tenth day of life (45 per cent) or longer (75 per cent affected). The most serious sequelae and the greatest variety of them also occurred in these groups.

Pathogenesis of Kernicterus.

By V. JIRSOVA, M. JANOVSKY and M. JIRSA (*Prague*).

The authors bring experimental support for the theory that the polarity of bilirubin is a decisive factor in the development of kernicterus.

They used taurobilirubin as a model for conjugated bilirubin and by giving it intravenously to newborn and adult rabbits showed that it had no effect on the pigmentation of the brain. Using a commercial nonpolar and unconjugated bilirubin, the solubility of which in lipids is well known, they succeeded in colouring the brain tissue.

Clinical observations confirm that high levels of direct-reacting bilirubin do not play any part in the development of kernicterus.

Hyperbilirubinaemia as the Cause of Early Cerebral Palsy.

By M. FARKOVA and MINARIKOVA (*Olomouc*).

In searching for infants damaged by hyperbilirubinaemia in cases of Rh or ABO isoimmunization, or in full-term and premature infants without any isoimmunization disorder, we drew the following conclusions:

In our material from the period 1954-1958 we did not find any signs of damage to the central nervous system by hyperbilirubinaemia in full-term infants without isoimmunization disorders. In infants with haemolytic disease who survived, and who had clear signs of damage to the C.N.S. in the acute stage, we found clinical improvement.

Developmental Abnormalities Causing Cerebral Palsy.

By K. BRACHFELD and J. SVATY (*Prague*).

The 21 children sent to the Second Children's Clinic in Prague in the past 10 years with a diagnosis of cerebral palsy were found to have various abnormalities. These included one case of arhinencephalia, 6 cases of agenesis of the corpus callosum, and 14 cases of agenesis of the septum pellucidum.

The "Spastic" Palsies of Childhood.

By V. PITHA and M. FISAROVA (*Pilsen*).

Spasticity is nowadays a more clearly defined clinical picture than it used to be. Among 160 "spastic" children (diplegias, hemiplegias etc.) 8 per cent could be described as really spastic; in the rest the hypertonus was of non-pyramidal type. Only 3 cases showed extrapyramidal hyperkinesias. It is therefore preferable to speak of "hypertonic palsies" of childhood, and to reserve the term "spasticity" for really spastic cases.

The Atonic Palsies of Childhood.

By M. FISAROVA and V. PITHA (*Pilsen*).

Atonic palsies are very common in children, and even in the fifth year of life 16 per cent of palsied children can be described as hypotonic. These include not only so-called "atonic" diplegias but also the hemiplegic cases whose frequency is about the same. The hypotonia is well marked at rest, while in the upright position some slight postural signs can be observed later in life.

In some flaccid newborn babies, nociceptive stimuli produce very brief fits of the decerebrate rigidity type, but the prognosis is good; the children may be a little retarded but they never become spastic or hypertonic.

Primary Simple Oligophrenia (Primary Amentia).

By V. PITHA and R. NESNIDALOVA (*Pilsen*).

The primary amentia described by Ford is very common, and about 50 very typical cases have been collected in 3 years. The term "primary amentia" is used for another syndrome in psychiatry and for this reason "primary simple oligophrenia" is preferred.

Defective Speech in Children.

By R. NESNIDALOVA and V. PITHA (*Pilsen*).

Dysphasic troubles are very rare in cerebral palsy. They were present in 6 of the 200 cerebral palsied children examined. The rest had troubles of articulation of different types. It should be borne in mind that sometimes (6 cases) well-developed speech can be lost for a time (2 months to some years). In these cases an organic cause was found, and the authors speak of the loss of initiation of speech.

Differential Diagnosis of Motor Mutism.

By G. GOLLNITZ (*Rostock-Gehlesheim, Eastern Germany*).

The term "motor deaf-mutism" is criticized. The term "dysphasia" is recommended for all types of speech disorder of central origin in children. Expressive dysphasia is caused by two well-known brain-pathology syndromes:

- (1) congenital suprabulbar paresis; and
- (2) orolingual apraxia.

Three clinical cases were described. A film demonstrating the two types of primary expressive dysphasia was shown.

Infantile Cerebral Palsy with Epileptic Fits.

By V. VOJTA and J. ODVARKOVA (*Prague*).

Here 103 cases are analysed according to types of motor disturbance, time of onset of the first fit, mental level, and severity of the epileptic progress. The first attack may occur as late as the end of the 4th year. Epilepsy is very severe in infants with cerebral palsy. The diplegic form seems to be the most benign. In intellectually normal children fits are not so serious. In children with the more severe degrees of oligophrenic syndrome, fits are difficult to cure. The most severe epilepsy is encountered in the hypotonic form of cerebral palsy.

Perinatal Encephalopathy, Oligophrenia and Epilepsy.

By K. SCHWARTZOVA (*Pilsen*).

63 epileptic oligophrenic children with perinatal encephalopathy have been followed clinically and by E.E.G. In profound oligophrenics the epilepsy appeared at an earlier age than in the mental defectives. In severe oligophrenics the epilepsy often ran a malignant course—i.e., the seizures became increasingly frequent or were resistant to therapy. The most frequent type of fits was grand mal, but the defectives suffered equally often from psychomotor and other temporal lobe seizures. Petit mal was relatively frequent among the defectives, and tonic, akinetic or salaam fits were relatively common in children with the more serious degrees of oligophrenia.

The degree of intellectual impairment mainly depends on the extent and severity of the cortical disturbances, whereas the presence and degree of behaviour disorders depend also on the presence of temporal lesions and the influence of environment.

Pachygyria, a Rare Malformation of the Brain.

By J. DVORACKOVA, J. LICHY and J. TINTERA (*Hradec Králové*).

A case of pachygyria is reported in a boy of 3½ years, the second child of an epileptic and mentally retarded woman. From the age of 6 months the child had epileptiform attacks and was retarded in his mental development. He had a spastic palsy of the lower extremities and died in hyperpyrexia after 14 days' unconsciousness.

At necropsy, pachygyria was found in the frontal and parietal lobes of the brain. Microscopically, impairment of the cytoarchitectonic structures was seen within the cortical areas affected with pachygyria. The impaired zones were characterized by 4 structural layers and numerous heterotopic islands of cortex in the white matter, in the form of ganglionic nodules and clusters and solitary nerve cells. This picture corresponds with the early stage, at 3 to 4 months of intrauterine life.

Orthopaedic Surgery in Cerebral Palsy.

By J. SLAVIK (*Prague*).

Of the 7 cerebral palsied children born in a year per 100,000 inhabitants, 3 can be benefited by orthopaedic surgery. Orthopaedic surgical procedures are indicated in mentally normal cases of cerebral palsy and those with rigidity or athetosis.

Bone surgery, including osteotomy and stabilisation operations, has proved highly successful, especially in athetoid cerebral palsy. Transposition of tendons resulted in failure in every case. So far, the long-term results of neurectomies have been bad. Tendon lengthenings and tenotomies have been more successful, but correction must be maintained by the use of plaster or braces until growth has been completed.

Motor Reeducation with the Help of Dancing.

By I. LESNY, J. PFEIFER and J. KRAUS (*Prague and Zeleznice*).

In 86 children with various forms of cerebral palsy music was used to help with motor reeducation. The children were taught to carry out simple dance steps like the samba and the polka. Compared with other rehabilitation methods used in the same institution this dance treatment gave better results. Improvement was seen in 90 per cent of cases.

The original purpose of the dance treatment was to treat extrapyramidal forms of cerebral palsy with incoordination, but improvement was also obtained in spastic forms. It is suggested that the treatment facilitates transmission in the connections of the acoustic and vestibulospinal pathways with the reticular formation.

Electrophysiological Evaluation of Guaiacol Glyceryl Ether in Spastic Cerebral Palsy.

By V. JANDA and V. SKORPIL (*Prague*).

Guaiacol glyceryl ether has an effect on spasticity similar to that of mephanesin, with less side-effects. To ascertain its action on spastic forms of cerebral palsy a number of signs were followed electromyographically before and after administration of the drug. The drug caused diminution of the Babinski reaction, ankle-jerk and ankle-clonus, improvement in the voluntary activity of the gastrocnemius during plantar flexion, and diminution of the pathological activity in the same muscle during dorsiflexion of the foot. The abdominal reflexes were unchanged.

It is evident that the drug has a favourable effect on spasticity. It greatly reduces polysynaptic activity and to a less extent monosynaptic activity, in which the muscle spindle plays a part. It appears, therefore, that the drug also acts on the gamma system.

The use of the drug in the rehabilitation of spastics can be recommended.

Effect of Small Doses of Reserpine on the Electromyograms and Chronaxies in Spastic Cerebral Palsy.

By I. LESNY and J. KRAUS (*Prague*).

An E.M.G. was done in 12 children with spastic forms of cerebral palsy. A dose of 0.1 mg. of reserpine per 10 k.g. body-weight was then given and another E.M.G. was performed 1 hour later. This invariably showed a more normal record in both frequency and amplitude. Chronaxies were examined in 20 children with spastic cerebral palsy before and after the same dose of reserpine. A shortening of chronaxies was observed in some cases, while in others a normal difference of antagonists reappeared.

Electroencephalography in Newborn Babies with Cerebral Damage Proved at Autopsy.

By M. ROSSLER (*Prague*).

In 9 newborn babies with cerebral damage the E.E.G. tracings during the perinatal period were abnormal. In 5 cases there were flat tracings without definite frequencies; in 3 cases there were tracings with frequencies and forms different from these of normal babies of the same age and birth-weight; and 1 case showed paroxysmal activity.

Hypermaturation Electroencephalograms

By I. LESNY and M. ROSSLER (*Prague*).

A study was made of 4 boys aged between 10 months and 4 years with delayed development of the central nervous system. Three of them also had a hypotonic form of cerebral palsy. These boys had surprising E.E.G.s: the records corresponded to those of a child 2-4 years older. Dominant rhythm of alpha frequency (8-10/sec) was present even in the records of boys of 10 and 18 months. This was found repeatedly. These observations are difficult to explain, but we believe that a hypermaturation E.E.G. can be a sign of an alteration in cerebral development.

Electroencephalography in Hemiplegic Cerebral Palsy

By I. LESNY (*Prague*).

E.E.G.s were done in 71 children with infantile hemiplegia, caused by cerebral hemiatrophy or porencephaly. Most of them also had epileptic fits. Group I (18 children) were in the age-group 1-3 years; group II (14 children) were aged 3-6 years; group III (16 children) were aged 6-10 years; and group IV (23 children) were aged 10-16 years. Only 3 records were normal. The abnormalities were more often generalised than focal. The focal E.E.G. abnormalities differed according to age. In group I they were present in 39 per cent of cases, in group II 21 per cent, in group III 12.5 per cent and in group IV 39 per cent of cases. Whereas in group I the focal abnormalities were mostly parieto-occipital, in group IV they were temporal. It seems that the E.E.G. tracing in the hemiplegic form of cerebral palsy, which represents a hemispherical lesion, is quite often focal in infancy. Between the age of 3 and 10 years it becomes generalised. In the prepubertal period it again becomes focal but shifts from the occipital to the temporal region.

Inborn Errors of Metabolism with Oligophrenia.

By M. BLEHOVA (*Prague*).

These errors are classified on the plan of Lippman, Wright and others. They may be divided into disturbances of protein, carbohydrate and lipid metabolism.

During a search for phenylketonurics, we found none among 5011 children in the schools of Prague, but we found 10 among the 2444 patients in hospitals for chronic sick and last year 3 more were added. Among the siblings of these patients we found 3 other cases, who are much better mentally, attending special schools.

In our country all children are kept under observation at medical centres from birth, and it should therefore be easy to examine every child for phenylketonuria 21 days after delivery, when phenylpyruvic acid can be found in the urine. In this way it should be possible to treat all affected children by diet from early infancy.

Developmental Anomalies in the Frontal Area of the Interhemispheric Fissure.

By L. ZDRAHAL and J. LICHY (*Hradec Králové*).

Clinical and pneumoencephalographic observations have been made in 67 cases of developmental anomalies in the frontal area of the interhemispheric fissure. Our cases included 5 agenesises of the corpus callosum; 8 agenesises of the septum pellucidum or cavum Vergae; 39 non-communicating dilatations of the cavum septi pellucidi (fifth ventricle) and 4 dilatations of the interventricular cistern.

The clinical pictures in these malformations are not specific but depend on the type of the anomaly and the age at which it appeared. In the agenesises, the syndromes of early infantile cerebral palsy with physical and mental retardation, epilepsy, and central paralysis are found most frequently. In the group of dilated cavum septi pellucidi and cavum Vergae the picture of early infantile cerebral palsy is much rarer, and epilepsy and headache are the most frequent signs.

Pneumoencephalography in Various Forms of Cerebral Palsy.

By J. DITTRICH, J. JIROUT and V. VLACH (*Prague*).

The pneumoencephalographic findings have been evaluated in 117 severe cases of cerebral palsy, 100 of them in children. No correlation has been found between the severity of the clinical findings and the P.E.G. changes. Nevertheless, some P.E.G. signs are found more often in certain forms of cerebral palsy. In the hemiplegic form the signs of contralateral cerebral atrophy were found most frequently. In the quadriplegic form the atrophy was bilateral. In diplegia the P.E.G. was often normal. The hypotonic and cerebellar forms commonly showed P.E.G. signs of brain-stem or cerebellar atrophy.

The value of P.E.G. in cerebral palsy lies in establishing a correct diagnosis, indicating suitable therapeutic measures, and interpreting the clinical picture on the basis of the anatomical changes it reveals.

Developmental Arrest and Developmental Regression.

By I. LESNY, J. DITTRICH and B. BEDNAR (*Prague*).

Five children were observed in whom development was arrested at the age of 9-12 months. In two of these cases central nervous system function went back to the earlier age of 4-6 months. The entire neurological examination corresponded to a baby of 4-6 months, though the children were in fact 3-5 years old. In 2 cases which died autopsy also showed regression of development (a 4-layer cerebral cortex). Thus not only can the development of the central nervous system be arrested in the postnatal period but its development can even recede, which seems to be contrary to developmental laws.

Pathogenesis of Harrison's Groove: its Occurrence in Infantile Cerebral Palsy.

By V. VOJTA (*Prague*).

The occurrence of Harrison's groove is ascribed to weakness and incoordination of the abdominal muscles, especially the lateral parts. Metabolic effects, in the broadest sense of the word, may contribute to its production. Most important are the influences of the muscles of the abdominal wall in the organisation of the stretch reflex.

Suitable exercises—making the affected children sit with retroverted head—can produce a co-ordinated contraction of the abdominal wall which pulls down the lower aperture of the chest and causes the Harrison's groove to disappear, since it is not fixed until after the 10th year.

Physical Treatment and Education.

At the end of the meeting three films were presented showing new methods of motor reeducation and physical treatment, including dancing, at the National Centre for the Treatment of Cerebral Palsy, Zelenice, Northern Bohemia, and at the Jedlicka Home for Crippled Children in Prague.

EDITORIALS

ABDOMINAL DECOMPRESSION IN LABOUR

DURING the uterine contractions of normal labour the oxygen supply to the foetus is temporarily reduced, but the signs of asphyxia are intermittent, and recovery is rapid and complete between contractions. Occasionally hypoxia may be severe and prolonged enough to cause irreversible changes in the brain or even foetal death. The presence of pre-eclamptic toxæmia, hypertension, diabetes and postmaturity predispose to foetal asphyxia during labour because of an underlying placental insufficiency. Hypoxic foetal deaths rise proportionately with the length of labour. Although forceps can be used to shorten the second stage of labour, no safe way, apart from Caesarean section, has been found for shortening the first stage. Oxytocin drips may shorten labour but they tend to cause prolonged and painful uterine contractions with an increased risk of foetal asphyxia. In Sweden, Malmström has perfected the vacuum extractor for application to the foetal head as an alternative to forceps or to stimulate contractions in cases of uterine inertia, but the use of this instrument as a means of shortening the first stage of labour appears to be limited. The discovery by Professor Heyns of Johannesburg that reducing the atmospheric pressure over the anterior abdominal wall shortens labour and relieves pain is therefore of the greatest importance.

In measuring the electrical potential of the uterus in labour Heyns found that when the abdominal muscles were paralysed with suxamethonium labour progressed faster than was expected. He ascribed this to the fact that the uterus could work at its best mechanical advantage by adopting a spherical shape unimpeded by the tense muscular abdominal wall. Other means of relaxing the abdominal walls, such as regional analgesia and hypnotism, were tried but abandoned in favour of decompression.

In his most recent experiments the patient was placed in a plastic bag of laminated polyvinyl chloride so as to contain the trunk from the groin to just above the breasts. Inside the bag was placed a rigid cage made of steel rods or fibre-glass which prevented the bag from collapsing over the abdomen or thorax when the pressure inside it was reduced. The patient sat down in a semi-reclining canvas chair with her legs and arms free outside the bag. A 70-litre-per-minute suction pump was attached to the bag and decompression was applied during contractions only, being controlled by the patient herself by means of a valve connected to a pressure gauge. It was found that pressures of 50-100 mm. mercury (1-2 pounds per square inch) below atmospheric pressure were readily produced over the abdomen and lower thorax beneath the incompressible cage within a fraction of a minute. There was slight impairment of respiration but this was offset by the remarkable relief from pain. The bag was closed by an airtight plastic zip which could be opened for listening to the foetal heart; no cases of foetal distress occurred in the bag. When the cervix was fully dilated the patient left her bag and was removed to the labour ward for routine delivery.

Of 100 primiparous women placed in the decompression bag early in labour 64 were delivered within six hours and 84 within ten hours. Most remarkable was the great relief from pain experienced during decompression; in 86 of the 100 women the relief was rated as excellent or very good.

Heyns suggests that decompression attains its results by allowing the uterus to become more spherical, so that the myometrial fibres of the upper uterine segment shorten more easily and thus dilate the cervix more quickly. This tendency of the uterus to become spherical is normally resisted by the tense abdominal muscle wall. Another

advantage of relaxing the anterior abdominal wall is that it leaves the axis of the uterus free to rotate forwards and thus improves the relation of the foetal axis to the brim of the pelvis. Heyns also noticed that the membranes ruptured late. This might be a result of the foetal head fitting the lower uterine segment better because of improvement in the direction of the foetal axis, or it might, as Heyns suggested, be a result of reduction in intra-amniotic pressure. In support of this second view no case of foetal distress was encountered in the 100 primiparae.

If decompression of the abdominal wall

does indeed shorten labour, the incidence of foetal distress from hypoxia can be expected to fall. And if there is at the same time a reduction of intramniotic pressure during uterine contractions, the incidence of intra-partum foetal asphyxia may be reduced still further. Even without these advantages to the foetus the pain relief obtained makes the technique one of the greatest value. Several decompression bags are now in use in this country, and it should not be long before we are presented with evidence to support Heyn's claim.

T. L. T. LEWIS

Conservatism in the Management of Foetal Distress

Unexplained foetal distress during the first stage of labour is a problem with which obstetricians are all too familiar. Norman Walker (Brit. Med. J. 1959, ii, 122), after reviewing the accepted criteria of foetal distress, draws attention to the different emphasis placed on these by various authorities; in particular, a varying amount of significance they attach to foetal tachycardia and bradycardia. After doing a pilot study of 350 cases, in which one or more of the accepted signs of foetal distress was present during labour, he carried out a controlled study of a further 350 patients in whom operative or conservative management of foetal distress was determined by random selection, provided there was no other indication for active treatment. From his comprehensive analysis of the results, the importance of a recognisable cause for the foetal distress is clearly seen. Thus cases in which signs of distress were accompanied by some obstetric complication had a foetal mortality four times greater than the otherwise uncomplicated cases (14.6 per cent; 3.6 per cent). The worst results were seen in cases associated with cephalo-pelvic dispro-

portion, especially when delivery was by forceps. Foetal distress unaccompanied by obstetric complications appeared to be less significant, and operative delivery in these cases seemed to give no better chance of foetal survival, the foetal results from spontaneous vaginal delivery, in fact, appeared to be better than those from caesarean section. However, it is not clear whether these two groups of patients were strictly comparable. Low forceps delivery, unlike the more difficult instrumental operations, was found to cause no increase in foetal mortality, thus underlining the value of this procedure for the early treatment of foetal distress during the second stage.

Walker makes out a good case for the conservative management of foetal distress in the absence of other complications. He considers that such distress should be . . . "an indication to search for the cause rather than a demand for dramatic action". Nonetheless, he admits that the birth of a live child is not necessarily the hallmark of good obstetrics, and the possibility of non-lethal cerebral damage in such cases has to be remembered. His emphasis on the import-

ance of foetal distress in association with other complications, especially with cephalopelvic disproportion, is a valuable reminder of the place of caesarean section in these cases. He concludes that meconium staining of the liquor is the only definite sign of foetal distress, the quantity of meconium being unimportant, and that "the behaviour of the foetal heart is of doubtful value . . . provided the umbilical circulation is not occluded". This last observation is of little

help clinically since, cord occlusion due to causes other than recognisable cord prolapse is not uncommon.

Information on the durations of labour before the onset of foetal distress, and on the incidence of post-maturity would have added to the value of this paper, but it is nevertheless an excellent contribution towards clear thinking on this difficult subject.

S. D. PERCHARD

Classification of Mental Retardation

It is easy to pick holes in classificatory systems, and mental deficiency is so full of syndromes of unknown, uncertain or multiple causation that its classification is particularly difficult. This fact clearly emerges in the "Manual on Terminology and Classification in Mental Retardation", issued as a supplement to the September, 1959, issue of the American Journal of Mental Deficiency, before its adoption at the 1960 Convention of the A.A.M.D. The manual follows predecessors issued in 1921, 1933, 1941 and 1957.

It is divided into five sections. The first, on Definition, gives a general formulation designed to distinguish between mental retardation and other disorders of behaviour. It represents the compromise "most acceptable to a majority of those many persons" reviewing the manual—that is, officers of the A.A.M.D. Mental retardation is defined as "subaverage general intellectual functioning (i.e., more than one standard deviation below normal) which originates during the developmental period (up to 16 years of age) and is associated with impairment in one or more of the following: (1) maturation, (2) learning, and (3) social adjustment." Maturation is defined as the rate of development of childhood social skills, and (most important) social interaction with age peers. Learning is defined as failure in the academic situation for schoolchildren. Social adjustment concerns a failure in social adequacy in terms of job-holding and con-

formity to community mores, and applies principally to adults. It is noted that there are still no adequate measures of the impaired functioning areas.

The second section of the manual sets out a revised medical classification into eight main groups. These comprise mental retardation associated with (1) infection, (2) intoxication, (3) trauma, (4) metabolism etc., (5) new growths, (6) unknown prenatal influences, (7) unknown structural changes and (8) unknown psychogenic changes. A decimal system is used to expand on the main groupings specifying syndromes, extra figures being added for additional secondary causes, such as impairment of special senses, motor function or psychiatric function. This section includes the outstanding feature of the manual—a terse and fairly accurate description of the majority of known syndromes.

In the third section, on Behavioural Classification, an attempt is made to describe behaviour along two dimensions—that of measured intelligence, such as Stanford Binet and Wechsler ratings; and that of adaptive (or social) behaviour, such as the Vineland scale. The best comment on the latter part is provided by the manual: with the adult it says, "there are few objective assessments available and the diagnostician must rely almost totally upon subjective judgements . . . and . . . strife and discord in family and community." The fourth section, on Statistical Reporting,

describes a hospital record system, using the proposed classification on a punch hole card, while the fifth section contains a useful glossary of terms for laymen.

The classification suggested here, like others, seems to be rigidly dependent on intellectual subnormality, for only those with a Stanford I.Q. of less than 83 or a Wechsler less than 84 are covered. It seems quite possible that those who might want to take part in or could be benefited by a training hospital programme would necessarily be excluded. In addition there is no mention of sexual perversions affecting dullards, although these provide some of the most important problems both inside and outside

hospital. It is possible to criticize minor points in the description of syndromes, and there is the usual criticism that an end product such as cerebral diplegia might be placed in any one of four groups. The classification barely notes psychogenic causes of defectiveness, despite the great deal of important research done recently. It seems fair to say that the manual provides valuable help with the classification of the severely subnormal, most of which are believed to have syndromes with pathological findings, but firmly passes by the main mass of psychogenically caused subnormals.

M. J. CRAFT

Mental Retardation Research Methods

The symposium on "Research Design and Methodology in Mental Retardation", held at Wood Schools, Pennsylvania, in May, 1959, marks an important stage in the emancipation of professional opinion from a policy of custodial care. (Amer. J. ment. Def. 1959, 64, 227-431.) As a visiting World Health Organisation consultant noted, the quality of American care for defectives varies as widely as do the States themselves, but the best hospitals have well-staffed and enthusiastic research departments as well as the necessary funds. This symposium seemed to be designed for the personnel of such departments.

The aspects of methodology reviewed by the writers—for the papers have been restyled to suit readers instead of listeners—include a historical survey by Cornfield, and a cheerful autobiographical approach by Margaret Mead. There is some practical discussion of the technique of extracting funds from research agencies and presenting a balance sheet at the end. Hobbs illustrates rather sadly the pressures exerted on a research worker to obtain a positive result,

for few American administrators seem to understand the value of a negative trial at the end of a contract. A variety of statistical approaches are detailed, together with the varied methods used in the analysis of data. Morton gives an excellent account of the problems involved in experimental design, while Masland and Gladwin review methodological approaches in the fields of aetiology and psychology which bring the student up to date from their recent outstanding analysis of current work on mental deficiency.

The American Association of Mental Deficiency is to be congratulated on the general standard of the twenty papers. They bring out very clearly the number of individual approaches existing in mental retardation research. The symposium contains a number of part reviews of different aspects of the subject, and the thread is sometimes difficult to follow. The symposium is therefore more suitable for the specialist research worker than for the general reader.

M. J. CRAFT

THREE PAPERS ON SPEECH DISORDERS

The International Journal of Phoniatrics has published* three useful papers prepared for the 11th Congress of the International Association of Logopedics and Phoniatrics held in London in August, 1959. Writing in German, with an English summary, Prof. Dr. R. Luchsinger, of Switzerland, surveys the inherited components from which spoken language develops, and the factors that may underlie some disorders of speech. He deals with the inheritance of fine motor control, intelligence, and language aptitude as effecting normal and pathological reactions. It is not easy to differentiate hereditary from environmental factors, but in the case of stammering there is a good deal of evidence that an undefined hereditary predisposition exists in many cases.

Prof. Lucio Croatto and Dr. Caterina Croatto-Martinolli, write in French (with English summary) from Italy, on the pathological conditions of the nasopharyngeal sphincter. They discuss the physiological functions of the soft palate in relation to respiration, sucking, swallowing, and particularly in sound-formation, including some information obtained by radiography and cineradiography. They report on the results of 140 surgical operations and tell us something of the role of speech therapy in cases of palatal deficiency.

The most interesting papers of the three, for Bulletin readers, is likely to be the one on "Defects of Articulation" by Dr. Muriel Morley, who practices speech therapy in England and is the author "The Development and Disorders of Speech in Childhood," and "Cleft-palate and Speech" In her summary Dr. Morley says: "Articulation is a learned neuromuscular skill based on physiological principles of motor learning. It involves effector and receptor processes which, through sensory feed-back, control the coordinations of muscular movements for articulation. Defects of articula-

tion occur when any of the processes involved are abnormal, or there is a failure to use the sounds of speech normally. Such defects have been considered in five groups, namely, (1) those secondary to a hearing defect; (2) dysarthria, where normal receptor processes can act only on and through a damaged motor mechanism; (3) apraxic dysarthria, where there appears to be a breakdown between normal receptor processes and a normal motor mechanism; (4) dyslalia, where there is a failure to use receptoreffector processes adequately, and (5) structural conditions of the organs of speech themselves.

Developmental dysarthria is often seen in children and adults with cerebral palsy, although many with severe motor disability due to pyramidal and extrapyramidal lesions may have normal or near normal speech. Marked dysarthria may be found in the absence of any other signs of cerebral damage. The term "isolated developmental dysarthria" has been used to describe some of these cases, and Dr. Worster Drought has described the condition on an aetiological basis as congenital suprabulbar paresis indicating that the lesion is presumably situated in the motor nerve pathway above the medulla.

Dr. Morley gives us a comprehensive exposition of all conditions giving rise to the wide variety of symptoms resulting from interference with the processes by which audible symbols are produced through movements of the organs of speech controlled by complex neuromuscular mechanisms. The aim of treatment in cases of dysarthria is to improve the mobility and coordination of muscle groups, and this may require the use of reflex inhibiting postures, such as those described by Bobath, when treating children with an associated general motor disability.

This paper can be strongly recommended to all concerned with patients needing specialised speech therapy, but it would not be suitable for parents.

LEONARD WILLMORE

Folia Phoniatrica, 1959, 11, nos. 1-3. The full proceedings of the Congress can be obtained from the Secretary, 46 Canonbury Square, London, N.1.

PSYCHOLOGICAL SERVICES FOR THE CEREBRAL PALSID

An Interdisciplinary Discussion in Miami

In an intensive postgraduate "workshop" lasting a week, held in Miami last year, specialists of various professional disciplines collaborated in discussing psychological services for the cerebral palsied. The University of Miami cooperated with the local Cerebral Palsy Rehabilitation Centre and the Vocational Rehabilitation Office of the Department of Health, Education and Welfare. Perhaps the chief interest of the papers presented lies in the picture they give of a total and interdisciplinary approach to the problems of helping the cerebral palsied to find their place in a normal society.

It is noteworthy that the Miami Cerebral Palsy Rehabilitation Centre extends its services to "all patients who have brain injury and brain damage which in some way alters their functioning, motor-wise or sensory-wise, or in a manner which makes their adjustment in society other than normal. Such a definition would include those individuals with behaviour problems on a brain damage basis, reading and learning difficulties, visual and auditory difficulties, mirror-writing phenomenon, etc." Psychologists in this country whose work extends beyond the immediate problems of cerebral palsy children have in recent years become vividly aware of the number of children who pass through the schools as normal, unhandicapped pupils, but who have learning difficulties and frustrations closely akin to those that characterise many cerebral palsy children. Enquiry into the histories of these children in birth and early infancy indicates the possibility that for many though not all of them difficulties may arise from some form of brain damage, and that they could have been helped and spared much misery if their teachers had known of their difficulties and of ways to help them.

The Miami Assessment Plan

In a paper on "Medical Evaluation and Diagnostic Assessment of the Cerebral Palsied", Dr. Engle briefly outlined the Miami centre's plan for assessment. This involves a very full examination by all the specialist services, neurological, orthopaedic, dental, audiological, visual and so on. He stressed the global nature of the problem and the need to combine with other specialists in formulating a plan for the care of the cerebral palsied, based on a total assessment. He emphasised the importance of the psychologist, as a person equipped to make the intellectual evaluation and also to determine the patient's adjustment to himself and his environment. "The patient's relationship to his world, his parents, his siblings, his neighbourhood, and his community must be understood. Educational planning, the patient's adjustment to an educational setting, his aspirations, the adequacy of his planning and many other factors must be explored in detail and worked through with the psychologist."

Dr. Engle described the function of the Department of Special Education in helping to formulate an educational programme. He stressed the need to interpret the patient to his family and vice versa and the importance of vocational habilitation if, as he said, "we are to treat the whole person and not just his limbs or his speech efficiency."

In these "workshops", when all the various evaluations have been completed, representatives from every department meet for final diagnosis and planning. The value of such meetings cannot be over-estimated. One of the regrettable aspects of work with cerebral palsy patients in England is the isolation of many workers from those of other professional disciplines. This limits

the usefulness of each member's contribution and leads to many contradictory decisions regarding plans for the patient. Dr. Engle stressed the need for constant re-evaluations and reminded his hearers that the patient will have cerebral palsy for the whole of his life. He will continue, therefore, to need help to adjust himself to new situations. The efficacy of the plans can be judged, and the progress or the need for a change of plan determined, only by re-evaluations in follow-ups.

Towards Independence

Constant emphasis was placed on the need to help cerebral palsy patients achieve an independent life. To this end vocational counselling plays a very important part. The use of tests of vocational aptitudes that have been standardised on the normal population tend to high-light things that the cerebral palsied cannot do. The aim of vocational counselling in the Miami department of Vocational Rehabilitation is to find the positive assets of cerebral palsy patients. For this they use a "work-sample technique", the main purpose of which is to ascertain the vocational potentials of cerebral palsy persons who have difficulties in finding and holding a job. The work of the project lasts for seven weeks, with evaluation sessions from 9.30 to 3.30 for five days a week. A very full assessment of the patient is made during the early weeks. This group of workers has found that not enough emphasis has previously been put on the perceptual abilities of these people; so that in the initial assessment period the ophthalmologist examines not only visual acuity but also continuity, depth perception, and peripheral vision. After the various examinations an interdisciplinary conference is held at which the case is reviewed and each department has a voice in future planning and recommendations. Through this work-sample technique the worker can be observed as a total, integrated individual functioning in a job setting that has all the on-the-job requirements involved in it. It is planned to stimulate as nearly as possible an actual job situation, and the differences in ability and capacity for each trainee on every job sample are carefully observed. This project is not only a means of assessment and testing but also offers opportuni-

ties for individuals whose work experiences have been limited to become familiar with tools and procedures. In this way it offers opportunities for observing the patient's capacity to profit from training. By observing the worker in a miniature "real life job situation" a great deal can be learnt about his capacity to work with other people and to adjust himself to the demands and problems that arise.

When the seven weeks' period is at an end the project evaluators and the United Cerebral Palsy Association staff meet in conference with representatives of community agencies. Out of this conference decisions can be taken. It may be that training is indicated or some kind of placement recommended either in competitive industry or in a sheltered workshop. This may seem an expensive and time-consuming procedure, but if it is thought of in terms of helping these handicapped people to become independent, with the consequent satisfaction and happiness which that can bring, it is infinitely worth while.

One of the disturbing factors in our own provision for the cerebral palsied is the unpreparedness with which we allow many children to move from the sheltered environment of a school for physically handicapped and/or cerebral palsy children to try to find their place in the adult world. Youth employment officers do their best to place them in jobs, but many of them have personality problems over and above their physical limitations which make it difficult for them to take their place in an unprotected environment.

Dr. Thomas Jefferson discussed these problems realistically and constructively. He stressed the need to reorient the concept of therapy, moving away from the confines of a doctor's consulting-room and the limits of a particular "school" of therapy in the direction of a "total therapy" approach. In this concept of "total therapy" a chance to work and a chance to practise interpersonal relationships play an important part. Dr. Jefferson believes that it is also of primary importance that all counselling and therapy should start with and include the patient's immediate family and should begin as early in life as possible. The family need help in their feelings towards their cerebral palsy

child and in learning to accept him realistically. Dr. Jefferson stressed also the importance of consistency and order in helping these children to realise their maximum potential. He urged the setting up of centres providing what he described as a "milieu therapy", through which the cerebral palsied patient can have the experience of a well-regulated environment where he can learn to solve simple problems within his capacity, in social situations that do not make too great a demand on him. In this way the patient will develop behaviour patterns which will eventually enable him to respond more adequately to the demands outside the Centre

and become better adjusted to home and community.

This is what the best of our own cerebral palsy schools are doing for their children. But this "workshop" emphasised the need for taking this care beyond the end of school life, through the difficult period of adolescence and into the world of grown-ups. This is perhaps one of the most serious gaps in our provision for the cerebral palsied. The survey of services for the cerebral palsied in Miami given to this postgraduate "workshop" offers some stimulating suggestions of ways for closing it.

J. M. WILLIAMS

PHENYLKETONURIA

The section on phenylketonuria was one of the highlights of the First International Medical Conference on Mental Retardation, held at Portland in July, 1959. To the pleasure of all present, Dr. A. Fölling of Norway, who made the original urine tests on patients in Oslo, took the chair on the day when the papers on this subject were presented. Two paediatricians, Dr. D. Y. Y. Hsia of the North Western University of Chicago, and Dr. R. S. Paine from Harvard, Boston, who spoke on enzyme defects and on protein and amino-acid metabolism, described phenylketonuria as originating as an enzyme abnormality and Dr. Hsia included it as one of the 70 or so known inborn errors of metabolism. This figure of 70 gave most of his audience cause to think, for it seems doubtful if many of those present could have mentioned more than a dozen or two. However, the number of diseases in which an inborn error can be considered significant is growing daily; the conditions mentioned included maple syrup disease and oasthouse disease, and another example of an amino-aciduria associated with mental abnormality has recently been described by Dent and his colleagues.

The diagnosis of this abnormality was approached by Dr. H. D. Grumer of Pine-land Hospital, Pownal, who described a tolerance test using a dose of 1.2 g. of p-hydroxypyruvic acid and the examination of 24-hourly urines, more especially for phenolic groupings. Two of the exhibits were related to the diagnosis of this condition in very young infants by direct examination of the napkin into which a small amount of urine had been passed. This technique was recommended as suitable for screening large numbers of very young children in an attempt to pick up the few isolated cases that exist.

The general chemical basis of phenylketonuria was discussed by Prof. J. N. Cumings, who briefly recalled the early research work on patients and then outlined the normal metabolic pathways of phenylalanine and tyrosine. It was clear that the probable block in the disease is an absence or gross diminution of phenylalanine hydroxylase, which is normally present in the liver. After referring to the older experimental work on phenylalanine and tyrosine, Professor Cumings mentioned the newer studies of tryptophane and indole com-

pounds. Here again there appears to be some enzyme abnormality, and it may be that the mental abnormality is connected with these indolic substances rather than with any excess of metabolites of the phenylalanine series.

Dr. H. A. Waisman, professor of paediatrics in Wisconsin, gave a valuable general review of the subject. He discussed his results with the phenylalanine tolerance test in normal subjects, in patients, and in possible carriers of the condition, and he made three special points. The test should be a 4-hour one, the L and not the DL form of phenylalanine should be given, and it is much better to follow tyrosine levels in the blood than those of phenylalanine. He also showed that the enzyme phenylalanine hydroxylase may not always be completely absent.

In the discussion on treatment Dr. Waisman mentioned that some patients showed too low a blood tyrosine and it is particularly in these patients that an addition of 1 oz.

(28 ml.) of milk a day is valuable. However, the blood level of phenylalanine should not rise above 2 mg. per 100 ml.

Dr. H. Bickel, from Marburg, Germany, recalled that he had first treated cases of this kind when working at Birmingham some 9 years ago, using a phenylalanine-free diet. In his experience it is no use treating patients over 8 years of age, and little good is likely to result if treatment is started at an age of 2 to 3 years; it is almost essential to begin therapy in infancy. It is often difficult to obtain the complete cooperation of the parents, but this is an essential feature of the treatment. Provided such adequate cooperation is obtained, and the treatment is started very soon after birth, remarkable improvement will be seen in many of the patients.

In a film of cases treated in England and Germany, Dr. Bickel demonstrated the improvement obtained in many of the children investigated.

J. N. CUMINGS

VIEWS FROM AUSTRALIA

"You get sympathy but not understanding, and you don't want sympathy" is a remark which must be in the minds of many mothers of cerebral palsied children. It is quoted by Dr. Elwyn Morey in the paper she read at the 3rd Annual Medical and Educational Conference of the Australian Cerebral Palsy Association. The conference was held at Perth in 1957 and the proceedings are now available in a well-produced 200-page book.

This quotation underlines some of the most interesting papers and discussions which took place. The Australian workers are obviously and rightly conscious of the

need to understand the whole of the cerebral palsied child, his relationships with his family and later his integration into society. In addition to understanding the child there is an equal need to understand the doubts and fears of parents and the attitudes of neighbours and employers. This emphasis on the whole person rather than his disability is, of course, well-recognised but tends to be submerged beneath the attentions of the many experts and specialists who make up the cerebral palsy team.

Dr. Morey's paper, on "the cerebral palsied child and his family", fittingly formed the inaugural session of the conference. Her varied experiences with mothers as individuals or in groups shows the value of an acute awareness of the emotional factors at work and demonstrates that, if well directed, parent groups have far more

*Proceedings of the Third Medical and Educational Conference of The Australian Cerebral Palsy Association, held in Perth, Western Australia on September 27-30, 1957. Published by the Spastic Welfare Association of Western Australia (Incorporated). Edited by Mr. J. T. Mitchell.

usefulness than mere exchange of "know-how". Her comments on fathers are wise and informative. Is it true that fathers tend to reject their cerebral palsied children after the first few years, or else set them impossibly high standards in an unconscious belief in the child's power to overcome his disabilities? Is the weakening of relationships between husband and wife seen sometimes in the family of the cerebral palsied child due to a lack of understanding of father's needs and attitudes? Wise and continuous counselling of parents is an important part of cerebral palsy care which is too often dropped after the "assessment" stage. Dr. Morey's emphasis on the difficulty mothers have in allowing their children to grow up leads on naturally to another great

concern of the Australian workers. Mr. Livesay, in discussing the problems of fitting the cerebral palsied into society says "emotional contractures may be more disabling than motor ones". Two sessions were devoted to this important problem.

Dr. Rickards, in the closing paper, points out three factors of great importance. Firstly that the individual with cerebral palsy is a frustrated person, secondly, that he has difficulties in communication; and thirdly, that he is still dependent at an age when he should be independent. The modern world does not take kindly to the frustrated, the isolated and the dependent, and therein lies a challenge which has yet to be fully met.

THOMAS E. OPPE

"JOURNAL OF CHILD PSYCHOLOGY AND PSYCHIATRY"

This new journal, the official organ of the Association of Child Psychology and Psychiatry, provides a needed forum for people interested in these subjects. The editors are C. B. Hindley, Elizabeth Irvine and Emanuel Miller and the journal is published in London and New York, by Pergamon Press. The subscription is 3 guineas a year for members of the Association, which includes membership, and 5 guineas a year for private people.

The first number covers a wide range of topics. An article by the Clarke's, reviewing the concept of "deprivation" and bringing it up to date, and Anthony and Scott's review of manic depressive illness in childhood, with a case presentation, are particularly useful. (Anthony and Scott's use of the phrase "extra-version of phantasy" to describe an acting out of phantasy is perhaps unfortunate).

Pringle and Bossio's study of prolonged separation provides data regarding the importance, in determining how successful the child's adjustment will be when he is placed outside the home, of parental (particularly maternal) attitudes towards

the child. Trained social workers concerned with the foster-care placement of children have for some time recognised the crucial difference between the parents' seeing placement as a way of doing something for their child or as a means of getting rid of the child. Some reference to this experience of others would have been desirable here.

Kanner's discussion of whether behavioural symptoms always indicate psychopathology ("psychopathy" in the table of contents) is a reminder against hasty judgments on the basis of symptoms. Those who consider the child as a total functioning being within a specific milieu are however unlikely to be misled in this way; Valentine's Penguin on this subject ("*The Normal Child and Some of His Abnormalities*", 1956) covered it fairly adequately and it is surprising that this book is not referred to.

There is a discussion by Miller on the origins of child psychiatry. Isaacs asks psychologists to consider the roles of truth and falsehood in the development of behaviour and makes no reference to the

psychoanalytic concept of reality testing. In a delightful article Winnicott considers the symbolic value of string for one of his patients. Hunter describes the mechanics of training a children's psychotherapist at the Tavistock Clinic.

The journal also contains some well done book reviews. The format and printing are attractive.

An editorial policy which, without sacrificing the aim of covering all aspects of child study, seeks to group the articles

in any one issue around one theme will enhance the journal's usefulness and intrinsic interest; concern with normal child behaviour will help to give it balance, and self-discipline by the contributors, with fearless editorial severity, will give the readers clearly and well written articles.

This number sets a high standard for both variety and quality, and we shall look forward to ready future ones.

L. SCHENDLER.

"CHILDREN"

The July-August number of this American "interdisciplinary journal for the professions serving children", published 6 times a year from the Children's Bureau of the Social Security Administration, Washington, D.C., contains four authoritative articles on various aspects of the Social Security Act as it applies to children. Katherine F. Lenroot writes on the origin of the social welfare provisions; Mildred Arnold on the growth of public child-welfare services; and Arthur J. Lesser on "Health Services—Accomplishments and Outlook". These are followed by an account of the care given to the mentally retarded in Sweden, by Elizabeth Boggs and Gösta Nordfors, and a paper entitled "No Homes of Their Own", by Wayne Vasey. There are the usual book notes, notices of international publications, news from "Here and There", and the come-backs from readers that are a particularly interesting and instructive feature of this journal. The inside cover shows us some "customers" waiting for the doctor at a well-child conference. These clinics for supervising the health of babies and preschool children have lately been springing up all over the U.S.A.

The Editor is Kathryn Close of the Children's Bureau, but the annual subscription (\$1.75 for non-American subscribers) should go to the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C.

E. Clayton-Jones

DEATH OF MME. STELLA ISABELLE ALBITRECCIA

Mme. Albitreccia, who was in charge of the Department of Speech and Special Education at the Poliomyelitis Centre, "Aide aux Enfants Paralysés", in the rue de la Croix Faubin, died in Paris on June 10. Her original work in the field of disorders of the body image and spatial orientation in relation to special education of children with neuro-central disorders was well known in many countries. She was formerly a professor at the Service of Disorders of Speech and Written and Spoken Language at the Faculty of Vienna. Since 1945 she had worked with Dr. Auguste Tournay on the problems of intelligence and pre-scholastic training in these children. Among other papers on her researches were those she read at the Société de Neuro-Psychiatric Infantile (Paris, 1954), at the World Congress of Occupational Therapy (Edinburgh, 1954), at the World Congress on the Physically Handicapped (London, 1957), and at the International Study Group on Child Neurology and Cerebral Palsy (Oxford, 1958), the last of which appeared in these columns (*Cerebral Palsy Bulletin*, Vol. 1, No. 4, 1958, pp. 12-17). Her brother is Dr. Victor Koerbel, of New York.

BOOKS—NEW AND NOT SO NEW

Speech and Brain Mechanisms

By WILDER PENFIELD and LAMAR ROBERTS

Reviewed by DR. R. G. WILLISON, M.R.C.P.E.

**A most distinguished and adventurous contribution to the literature on speech,
written for laymen.**

This book contains the results of ten years' work on speech mechanisms done by Dr. Wilder Penfield and Dr. Lamar Roberts at the Montreal Neurological Institute. The material suitable for a general audience was presented in the 1956 Vanuxem Lectures. (A list of the lecturers is included in the book—a page of names distinguished in many fields of literature and science.) Consequently the monograph as it appears now will have considerable interest for psychologists and speech therapists as well as neurophysiologists and clinicians. Six chapters, including an introduction and epilogue, are written by Dr. Penfield and five by Dr. Roberts.

The material for study is derived from craniotomies performed to relieve focal cerebral seizures; 273 on the dominant hemisphere and an equal number on the non-dominant side were accompanied by a special series of speech examinations in 72 of the patients, by Dr. Roberts.

Early on, Dr. Penfield says that "if there is a frontier between mind and brain, it is crossed twice by each utterance during a

lecture". But he adds the warning to the physiologist that "if he should state that nerve impulses moving in certain patterns are one and the same thing as mind, he accomplishes little for his future work except to deprive himself of a useful working terminology".

The second and third chapters are a discussion of the current thought on the physiology of the central nervous system. The discussion on the centrencephalic system is very clearly written and the definition enlarged by Professor Jasper's suggestion that it should include not only "integration of the function of the hemisphere but also integration of varied specific functions from different parts of one hemisphere". This useful addition takes us some way towards visualizing a central system which can both activate and select from cortical activity. After all, human behaviour appropriate to the environment must be highly selected from a number of choices evoked by sensation.

There is a short review of the physiological anatomy of the cortex and a summary of the work on cortical stimulation.

Some of this discussion brings to the fore the considerable difficulties in terminology and orientation which beset any discussion

W. Penfield and L. Roberts. Princeton University Press. London: Oxford University Press. 1959, pp. 286, 35s.

of cerebral activity. For instance, apropos of the psychological responses to stimulation, Dr. Penfield says "It is obvious that the mechanism of these psychological responses is of an order very different from that of the motor or sensory responses. The patient recognizes these experiences from his past as authentic . . . When the motor convolution is stimulated . . . he may be surprised to hear himself vocalizing, but he never has the impression that he has willed himself to do those things." In fact there is nothing to suggest that the mechanism of the cortex in terms of cells differs from place to place or indeed that electrical stimulation by the usual methods varies fundamentally in its effects in different regions. Connections are widely different in different regions and may be responsible for local differences in function. If we accept that neurones can conduct and in some way store, then it must be the pattern of arrangement and activity which determines the overall function of a group. It is fortunate for us that fragments of function can be elicited by electrical stimulation and by epilepsy, and Dr. Penfield has made outstanding use of these observations in localization studies. It is presumed that these fragments hint at the function of portions of brain but they tell us little about mechanism. Further on, Dr. Penfield makes it clear that positive electrical responses appear more readily when stimulation is introduced near an epileptogenic area. This shows how much electrical effects may depend on the state of activity of the underlying brain.

From this comprehensive historical review of the pathology of speech, Dr. Roberts concludes that "despite a century of study, the mechanisms of speech and aphasia remain as challenging problems." It is interesting to consider some of the possible reasons for this situation. Speech is distinct from many other forms of learning, in that the sounds and words which comprise it are part of a language handed on through generations of a community. The words are not generated by the individual but are learnt as having relation to virtually all forms of sensory and motor experience. In other words, complex patterns of sounds and concepts already preformed are absorbed by the speech mechanisms. It would perhaps

be surprising if there was precise anatomical localization in the cerebral mechanisms. The wide extent of human vocabulary however shows how precise is the physiological resultant.

The review of the literature and the analysis of the cases concerned with dominance are most valuable. An important finding is that, leaving out of consideration cases with cerebral injury before the age of two years, there is no significant difference in the frequency of aphasia following a right-sided operation in the right- and left-handed. Before the age of two years the right hemisphere may subserve speech if the left hemisphere is damaged. This is about the strongest evidence available that the brain becomes progressively less physiologically homogenous with age. Whether this loss of functional lability is a product of ageing, or of learning, we do not know. The study of speech however goes some way to indicate that much of the brain can only learn once in a lifetime. Dr. Penfield discusses this idea more fully in the last chapter.

The early chapters do much to place in perspective the operative findings which are the basis of this monograph. Dr. Penfield fully describes the techniques of cortical excisions near speech areas and mapping by means of electrical interference. Mapping depends on the finding that aphasic arrest of speech caused by electrical stimulation occurs in the hemisphere dominant for speech. Such an effect has never been evoked in the non-dominant hemisphere. It is demonstrated by means of a naming test in which the patient is shown a succession of card pictures which he is required to name, by saying "that is a dog", and so on. The phenomenon of aphasic arrest is lateralized, as opposed to vocalization which may occur during stimulation of either hemisphere. As far as dominance is concerned, the conclusion is that the left hemisphere is usually dominant, regardless of handedness, if those with cerebral injuries sustained in early life are excluded. As to localization within the left hemisphere, interference with speech is produced by stimulation of Broca's area, the supplementary motor area, and the posterior temporoparietal area. These areas are anatomo-

mically described and illustrated in the text.

The other main category of evidence is derived from the study of cortical excision. Dr. Roberts reiterates the important view that tumour material examined at autopsy is unsuitable for localization studies. Displacement of brain by growth and involvement of subcortical structures by invasion or pressure make identification of both normal and damaged areas too uncertain.

In this series aphasia occurred immediately after operation on the left hemisphere in 22 of 273 operations and did not occur in right-sided operations. Of these 22, 13 had no tumour. There is some admitted difficulty in drawing conclusions from any case in which the parts of brain removed are abnormal. Nevertheless certain of Dr. Roberts' general statements seem justified. The first relates to pre-existing damage, and says that limited excision of a previously damaged area may be followed by only transient dysphasia, provided the remaining brain functions normally. The order of importance for speech appears to be the posterior temporoparietal area, Broca's area and the supplementary motor area. Persistent dysphasia may occur during abnormal function or with extensive destruction of the left hemisphere.

The last proposition derived from the study of excision relates to the physiology of normal speech and leads again to difficulties both of theory and terminology. Dr. Roberts writes:

"One of the chief things that has retarded progress is the acceptance of such a concept as that which states that perception is divided into: first, conscious perception of sensory impressions, and second, linkage of content of perception with other images—a process which can be interrupted at one or the other level by a lesion".

He decries the term *agnosia*, particularly when subdivided into visual verbal, visual literal, etc. and goes on:

"there is not a single case in the literature of visual verbal *agnosia* without other defects, together with the ability to recognise some word at some time if the examination is detailed enough".

He then proposes that:

"comprehension of speech occurs after impulses have been received in the higher brain stem and both cortical auditory areas, and during interaction between the higher brain stem and the left hemisphere. Following interaction between the higher brain stem and the left hemisphere,

impulses pass to both cortical motor areas and thence to the final common pathway to those muscles used in speech".

This proposition seems to carry both the implications of a sequential process of perception and of the possibility of interruption of separate pathways by local lesions. In fact, the main conclusion which does emerge is that the anatomical areas concerned with speech are each concerned with most of its aspects. The functional precision of speech must therefore lie within smaller physiological units which are not separable in the gross anatomical sense. It appears also that the authors have gone far in showing that speech is very much a "one channel" function with little duplication of essential mechanisms. This allows the reservation that these mechanisms may be situated in an unusual anatomical site if damage to the brain occurs early enough in life. Furthermore, it seems clear that local destruction and epilepsy make less brain available for the recovery of speech function after surgery.

Dr. Penfield finishes the book with two chapters, a Concluding Discussion and an Epilogue. In the Discussion he elaborates his ideas on the importance of the subcortical connections in the coordination of the functions of the cortical zones. He discusses the removal of the convolutions which surround the speech areas. These removals do not produce aphasia, and he concludes that the integration of the speech areas must depend on their connection with some common subcortical zone. He discusses the evidence provided by the occurrence of aphasia in lesions of the thalamus and the thalamo-cortical connections and presents the anatomical data germane to his hypothesis. In a description of the psychology of speech he is well aware that the neuro-anatomical and the psychological lines of thought are parallel. He comes nearest to linking the two when he uses the terms "ganglionic equivalent of a word" and "ganglionic equivalent of a concept". At present we have no model and no vision of these terms. He touches on the problem of self-consciousness:

"the awareness, that must be present before speaking and the understanding of speech is made possible, depends upon the passage of neuronal potentials through the multiform circuits of a centrencephalic system. The infinite

complexity of that system in the higher brain stem and its connections with the cortex are far beyond our present capacity to visualize".

With the background of this extensive anatomical study of speech the findings "may point out a direction for psychological thought"; and it will be evident that many workers will find those volume a most stimulating source of ideas founded on anatomical investigation.

The Epilogue is a discussion on language and learning. It should be of great interest to those interested in the development of speech and language in the young.

The authors' aim of presenting the evidence from operation study in monograph form, with a review of the literature, is admirably fulfilled. Their intention to make a clear statement of the neurophysiology of language and to locate its mechanism is also carried out within the limits of present knowledge. When lack of knowledge forces them to hypothesis we are grateful for such a clear and constructive formulation of the problems.

This is a most distinguished and adventurous contribution to the literature on speech.

The volume is well produced. The illustrations, bibliography and index are excellent.

R. G. WILLISON

Outline of Human Genetics

L. S. PENROSE. *London*: Heinemann. 1959, pp. 146, 12s. 6d.

For years generations of doctors have bewailed the increasing specialization of their successors in the medical schools and the narrowness of the basic education permitted to those schoolboys who boldly choose medicine as a profession. Nevertheless the deans of medical schools, or whoever select medical students, may well be right, for nothing so ill-equips a man for the prolonged study of medical textbooks as a lack of acquaintance with the classics of his own or any other language. Fortunately there are islets in this formidable ocean and Professor Penrose has provided one more.

It was the year of the general strike when, sitting in a deck-chair overlooking the

tennis courts at school, I made the acquaintance of Abbé Mendel and the delightful neatness of his observations. While the hearties went off to be special constables and trauunguards, we who had examinations to take talked provocatively of "lock-outs" and read widely. Ever since then I have hardly dared to expose my hard-won knowledge to the gales of more modern experience gained since 1926, and the few times I tried to read textbooks on the subject showed how wise I have been.

Here in plain workmanlike English, with his meanings never obscured by a proliferation of words, Professor Penrose lays down the foundations for an old foggy like myself as well as for a student of science, or art, or classics, or anyone who wants to know something about a subject of increasing importance and relevant to so many of our problems. When so many people talk nonsense about eugenics, this short book, well written, well printed and well illustrated, can show what is or is not possible. It should be required reading for all literate Members of Parliament. Just to show that I understand what the author intended, I believe that on page 106 he would have written, of the female carrier of haemophilia, that the risk to the sons of half her daughters was one in two, rather than "for her daughters' sons, one in four", had he not preferred the clearer statement.

DENIS PIRRIE

Speech and the Development of Mental Processes in the Child.

A. R. LURIA and F. I. YUDOVICH. *Translated by J. Simon. London*: Staples Press. 1959, pp. 126, 15s.

In this interesting report of an experiment done some time ago Professor Luria and his colleague describe a systematic study of the development of speech in a pair of identical twins. These twins, who communicated by their own private language, were deprived of each other's company for a short time and the resulting development of their normal Russian speech was recorded. This unique situation provided the investigators with an

opportunity to study all stages in the development of a language, which reached normal children's level for their age in a very short time. The general hypothesis that the growth of such a language would introduce important new peculiarities in the structure of the children's thinking was confirmed. After three months, play activity was frequently accompanied by speech, while formerly it had not been. Constructive activity improved and projects for play were formulated verbally in advance.

So far as this reviewer has been able to interpret the report, special training of one twin in language habits did result in some differences. The superiority resulting from the training, however, was confined to speech and operations connected with it. The importance of such training activity is emphasised by the authors. As they say (p.110): "The process of selective generalisation has a developed, objective character and transcends the limits of direct perception of the objects". On 119 the authors observe: "After ten months of the experiment both twins developed full-value practical speech activity as a result of which there was a perceptible reorganisation of their intellectual processes. But only one of them, Twin A, who had undergone continuous systematic exercises in speech, developed a 'theoretical attitude' towards speech proper to his age".

The book contains a number of notes which are very useful in tracing sources. In an introduction, Professor Zangwill welcomes the book as a "noteworthy contribution to the experimental psychology of language".

N. O'CONNOR

The Natural History of Cerebral Palsy

BRONSON CROTHERS and RICHMOND S. PAINE. *Cambridge, Mass.*: Harvard University Press. *London*: Oxford University Press. 1959, pp. 299. 32s. 6d.

This important book is based on the authors' huge experience in the Children's

Medical Center, Boston, and more particularly on their attempt to follow-up 1821 patients seen in the hospital or privately between 1930 and 1950. They succeeded in contacting 1401 of these patients and re-examined 655—some more than 20 years after their previous assessment.

As the authors point out, their series is probably not representative of the cerebral palsy sufferers in the community. Nevertheless the book would still have been extremely valuable if it had consisted only of a bare account of the clinical findings of such a carefully studies series, described with such a wealth of beautifully presented statistics. In fact it is much the most detailed that has yet been done. The authors have tried to show what cerebral palsy does to children—how it affects their physical, intellectual and emotional development, and how it affects their families. Such a task could have been undertaken only by people with the sort of insight, mature wisdom and great common sense for which the late Dr. Bronson Crothers was renowned.

Some criticisms must be made. The sections on classification, aetiology and pathology might well have been expanded to provide more comprehensive accounts of these aspects of cerebral palsy. Though they contain pearls of great value, the chapters on "Emotional Status in Adolescence and Early Adult Life" and "The Effect on the Family" are somewhat unnecessarily diffuse and anecdotal. The chapter on "Clinics for Cerebral Palsy" might have been placed more suitably with the others devoted to treatment, and could, indeed, have been combined with them. Since psychological aspects of cerebral palsy are the subject of a companion volume by Dr. Edith Meyer Taylor, it is understandable that the chapters on "Intelligence" and "Education" should be rather brief. On the other hand, there seems no good reason why there should not have been a much fuller account of the actual techniques of examination used by the authors in patients of different ages. This would have helped particularly in making clear the basis of the clinical classification of patients, 13 per cent of whom were placed in a category of "mixed cerebral palsy".

It seems odd to find a full chapter on kernicterus, which is distinguished from other extrapyramidal cerebral palsies, when such scant attention is paid to ataxic syndromes. The word "hyrocephalus" does not even appear in the rather inadequate index.

In spite of these minor shortcomings, the book is a major contribution to the literature on cerebral palsy, not only for the findings it describes but also for the wide understanding it reveals of the human problems involved and the advice it gives on their management. Perhaps the best tribute that one can pay is to say one wishes that it had been longer and had gone into more detail and given an even fuller account of the authors' methods of examination, their findings and their interpretations of other work.

It is a pleasure to note that the volume is well printed, has few typographical errors, is extremely well illustrated and is comfortable to hold.

T. T. S. INGRAM

Advice to the Expectant Mother

PROF. F. J. BROWNE and PROF. J. C. McLURE BROWNE. *London & Edinburgh: Livingstone. 1959, pp. 48, 1s.*

This edition of "Advice to the Expectant Mother" has been completely revised by the authors. It stands as a fine example of simplification and condensation and remains remarkably comprehensive for such a humble publication—it is but a paper-backed booklet. Nevertheless the eminent authors mention in their preface a criticism that there was too much detail in previous editions. Such criticism is hard to sustain in the face of, for example, the account of all aspects of toxæmia in less than 2½ pages.

There is a chapter entitled "Hints on Breast Feeding and Care of Baby", but no reference to artificial feeding appears anywhere in the booklet. This seems strange in view of the extent to which artificial feeding

is carried out in the United Kingdom—at least when Mother returns home!

This excellent publication is firmly established both here and abroad. It carries, of course, a most useful index.

W. A. BULLEN

Before and After Childbirth

JANE MADDERS. *London & Edinburgh: Livingstone. second ed. 1959, pp. 30, 3s.*

The first edition of this booklet established it as one of the most practical and useful of the growing number of publications for the expectant mother. It is essentially a "picture book", with a couple of large clear illustrations, mostly photographic, to a page, each accompanied by a few direct and authoritative though simple lines of text. It is difficult to suggest an alternative, but the fact that most of the obviously pregnant patients are depicted in bathing costumes—some briefer than others—strikes a faintly bizarre note.

This booklet must have proved a boon and a blessing to many women, and even Father manages to get into the picture on the last page!

W. A. BULLEN

Having a Baby

J. F. ROBINSON. *London & Edinburgh: Livingstone. 1958, second ed. pp. 100, 6s. 6d.*

The comprehensive nature of this book comes as a surprise. In just over 100 pages the author covers in a delightfully straightforward, but authoritative way, very many aspects of childbirth. In fact, the book might better be titled as "Having Babies". The chapters on "Contraception" and "Sterility" for example, set out shortly and authoritatively much more than is implied by the original title. The author claims in the preface that he set out to give "simply plain facts of married life".

W. A. BULLEN

ABSTRACTS

IN COLLABORATION WITH "Abstracts of World Medicine", published by the British Medical Association, and "World-Wide Abstracts of General Medicine", published by the Excerpta Medica Foundation.

Effects of Obstetrical Hazards on the Development of the Child

DUNCAN E. REID. *Journal of Obstetrics and Gynaecology of the British Empire* 1959. 66, 709-720.

Birth injuries are now known to be primarily due to intrauterine asphyxia rather than to trauma. Hence the aetiological factors can be reclassified as follows:

(1) A decrease in the rate and amount of foetal-placental blood-flow through cord compression.

(2) A decrease in the maternal placental flow of blood, due, for example to hypotension in spinal anaesthesia, to tetanic uterine contraction in prolonged labour, to improper use of oxytocic drugs, or to abruptio placentae or placenta praevia.

(3) A decrease in the exchange of respiratory gases across the trophoblastic membrane, perhaps due to fibrinoid deposition, placental infarction or ageing. The infants who die in utero in cases of pregnancy toxæmia may sometimes be suffering from an interference with placental membrane exchange.

(4) A decrease in the maternal alveolar exchange of respiratory gases. This must be extreme before it affects the foetus, for severe cardiac failure in the mother often does not damage the child.

For clinical purposes, intrauterine asphyxia may be classed as acute or chronic, the latter including placental insufficiency, whatever the cause. In infants who survive after chronic asphyxia the central nervous system is rarely damaged, whereas those delivered under acute episodes of anoxia, as in cases of late pregnancy bleeding or compression of the cord during delivery, are more likely to develop nervous sequelae.

Among factors which may have remote effects on the child is kernicterus, which is

not caused only by blood-incompatibility. Some believe it can be caused by drugs given to the mother in pregnancy or to the infant at birth. The use of vitamin K has been questioned because it is known experimentally to produce icterus. All forms of maternal therapy should be constantly watched as possible causes of harm to the foetus or newborn.

The extensive experiments done on the production of malformations by teratogenic agents, including irradiation, anoxia, nutritional deficiency or viral infection, have demonstrated that congenital defects appear only after a prodigious insult to the embryo.

The comparative influence of genetic and environmental factors has been difficult to determine. This is well illustrated in mongolism, which is generally attributed to various environmental disturbances but has now been shown to have a genetic origin, or at least to be associated with an extra chromosome.

Many viruses have been suspected, but only that of rubella can be implicated in causing a grossly defective child. Between 15 per cent and 20 per cent of women who contract rubella in the first 8 weeks of pregnancy give birth to infants with marked physical and mental defects.

There is no proof that maternal malnutrition produces abnormalities in the human foetus.

The potential harm which ionizing radiation may do to biological processes is especially to be thought of during pregnancy, for foetal tissues are particularly vulnerable to irradiation. X-ray examinations of the chest, and pyelography restricted to 3 plates with

the foetus screened, are probably fully justified, and it seems reasonable to permit X-ray pelvimetry if it is not repeated.

As to bleeding late in pregnancy, the tendency now is to treat patients suspected of placenta praevia expectantly until 5 weeks from term, when the foetus is mature enough to survive.

The obstetrician would like to know how much asphyxia the foetus can endure without suffering irreversible damage to its central nervous system. Up to now the only gauge of the foetus's condition in utero is the state of its cardiac function. Marked foetal bradycardia and irregularity are found in maternal syncope and when uterine contractions are frequent and strong. E.E.G. studies of the foetal heart have shown no abnormalities in toxæmia of pregnancy and hypertensive disease, so this test will not predict death of the foetus in utero, a threat always present in these conditions. Chemical determinations of the cord blood have only a limited value and are unsuitable for the routine clinical assessment of the infant's physical state at birth: reliance must therefore be placed on the physical signs and symptoms which the foetus exhibits at delivery.

Labour prolonged beyond 20 hours is a major source of foetal hypoxic damage. Uterine inertia should be treated energetically and relatively early with uterine stimulants, and labour should not be permitted to last longer than 30—40 hours.

Most authorities now hold that foetal mortality and morbidity are less with conduction anaesthesia than with general anaesthetics. The less complicated cases are best delivered under spinal or local block anaesthesia, and the more complicated under the inhalation type. Excessive medication for pain is avoided by an awareness of the obstetrical problems and reasonable anticipation of the time of delivery, so that medication is given according to the patient's needs rather than "by the clock".

E. Clayton-Jones

(Based on an Excerpta Medica abstract.)

Diagnostic and Therapeutic Problems Raised by Hip Involvement in Children with Cerebral Motor Disabilities.

M. HYON and G. TARDIEU. *La Semaine des Hopitaux* 1959. 35, 2695-2700.

A systematic clinical and X-ray examination of the hips was done in the 83 severely handicapped children of 2-10 years being reeducated at the Garches Centre. The hip abnormalities were classified into 3 groups: (1) Dysplasia, which may affect the acetabulum, the obliquity of whose roof is increased; the head, which is irregular and slightly off-centre; or the neck (coxa valga or vara). (2) Subluxation, where the head is clearly off-centre and raised but still has some contact with the acetabulum. (3) Dislocation, where the head has lost contact with the acetabulum.

In 48 children the hips were normal. The 35 with abnormal hips included 3 bilateral dislocations. In 10 cases there was subluxation, including one severe unilateral subluxation with dysplasia of the opposite hip. There were 22 children with dysplastic hips, usually bilateral but often worse on one side, always involving the neck (20 coxa valga and 2 coxa vara) and often the head and acetabulum also.

Why were the hips so often abnormal? Congenital predisposition seems probable in the 4 severe cases which had had bony injuries. In the other cases, muscular and static troubles account for the abnormalities. Several important points emerge. The first was that 32 of the 35 children with abnormal hips could not walk, compared with only 15 of the 48 children with normal hips. The type of muscular injuries is also important. Hips are more often abnormal among the spastic children. Stiffness of the adductors is almost always present.

As regards treatment, in one case of bilateral dislocation closed reduction on one side and open reduction on the other led to a good result after 15 months. The two other children with dislocation did not seem likely to profit from reduction, so no operation was attempted. In cases of mild subluxation or dysplasia abduction is maintained with long Phelps braces and the children learn to walk

under periodic radiological control. When abduction cannot be obtained, adductor tenotomy can be useful, this being eventually completed by lowering the insertion of gluteus medius. The best way to prevent hip abnormalities seems to be to teach those children to walk as soon as possible.

The points to be noted are: (1) the frequency of hip abnormalities; (2) the danger of muscular stiffness, especially adductor spasticity; (3) the importance of standing and walking; and (4) the good effects of long Phelps braces, allowing hip abduction and standing.

G. Tardieu

Hyperbilirubinemia in Premature Infants: Follow-up Study

C. A. KOCH, D. V. JONES, M. S. DINE and E. A. WAGNER. *Journal of Pediatrics*, 1959. 55, 23-29.

At Cincinnati General Hospital serial estimations of the serum bilirubin level were carried out in 1953 by one of the authors (Dine) in 100 consecutive newborn premature infants who neither suffered from sepsis nor showed any evidence of isoimmunization. The infants were observed carefully for the first few weeks of life and then re-examined between 2 and 3 years later. Many of these infants had received up to 10 mg. of vitamin K daily, a not unusual procedure in 1953. Of the 8 infants dying in the neonatal period 2 were found at necropsy to have kernicterus; in both these cases the serum bilirubin level rose above 20 mg. per 100 ml., whereas in the other 6 cases the bilirubin value remained below this level. In the present follow-up study 49 survivors of the original 100 were re-examined in 1955 and 1956. Neuromuscular abnormalities attributable to kernicterus were found in 5 patients, 4 being mentally retarded and either spastic or athetoid, while the remaining child was deaf and walked with a wide-based gait. In 31 of the 49 infants the maximum serum bilirubin level at birth was below 20 mg. per 100ml. and all these infants were normal, but of 14 in whom this

level had been between 20 and 30 mg. per 100 ml. 2 were affected, while of the 4 infants with a serum bilirubin level above 30 mg. per 100 ml. 3 were affected. There was not clear relationship between the later development of kernicterus and the vigour or weight of the infant at birth.

The 5 abnormal infants did not differ noticeably from the normal children in the early weeks of life; thus none had convulsions or hyper-irritability, and the 2 with the highest serum bilirubin levels were vigorous and active and took their feeds well. Careful observation over several years is therefore necessary to detect the true incidence of brain damage. The authors conclude that a high serum bilirubin level is the most important aetiological factor in kernicterus and advocate that when this level rises above 20 mg. per 100 ml. exchange blood transfusion should be given.

F. P. Hudson

On the Motor Deficit in Congenital Bilateral Athetosis

T. E. TWITCHELL. *Journal of Nervous and Mental Disease* Aug. 1959. 129, 105-132.

The author has studied at the Joseph P. Kennedy Jr. Memorial Hospital, Brighton, Massachusetts, the various movements made by children aged between 3 and 14 years who were suffering from bilateral or generalized athetosis not accompanied by spasticity or cerebellar symptoms. Detailed analyses of these movements, together with frequent references to the literature, leads to the conclusion that they are responses to primitive reflexes.

The main reflex actions are (1) the "avoiding" movement, which causes the high-stepping gait and the turning away of the head and trunk from a desired object; (2) the asymmetric tonic neck reflex, which results in flexion of the arm used in grasping the object and extension of the opposite arm; and (3) the labyrinthine and righting reflexes, which produce abduction of the limbs and the typical "associated" and "mass" muscle movements. He also found that the grasp reflex was really a more

primitive traction reflex, accompanied by flexion of the wrist—hence the weak grip. The author points out that all these reflexes are present in the normal infant, but are gradually suppressed as voluntary movements develop. In the athetoid child there is a failure of maturation of motor function, but as he found that some children improved with time he suggests that later maturation does occur. Unlike the findings in spasticity it was noted that resistance to passive movements affected both flexor and extensor muscles and was due to constant reflex activity; this activity also prevented sustained muscle contraction and so resulted in muscular weakness.

(The original paper should be of value to those dealing with athetoid children since the author there demonstrates how these reflex actions can be brought into play to assist voluntary movements.)

Janet Q. Ballantine

Congenital Anomalies Associated with Cerebral Palsy and Mental Retardation

R. S. ILLINGWORTH. *Archives of Disease in Childhood* June 1959. 34, 228-230.

The incidence of associated congenital anomalies in an unselected group of 278 children with cerebral palsy of pre-natal or natal origin is compared with that in 386 children with mental retardation of pre-natal or natal origin, excluding mongols. The incidence of associated anomalies was 6.8 per cent in the children with cerebral palsy and 26.4 per cent in the children with mental retardation alone. The high incidence of cleft palate and congenital heart disease in the mentally retarded group is noted. The implications of these findings are discussed. —(Author's summary.)

The Diagnosis of Mental Retardation in Infancy: a Follow-up Study

R. S. ILLINGWORTH and L. B. BIRCH. *Archives of Disease in Childhood* June 1959. 34, 269-273.

In this paper from the University of Sheffield a follow-up study is reported of a

series of 101 children who had shown evidence of mental retardation before the age of 2 years, the series being unselected except that cases of mongolism, cretinism, hydrocephalus, and anencephaly were excluded. The original diagnosis was made on the basis of the results of simple tests from the Gesell schedules, attention also being paid to the developmental history. The children were re-examined when aged 5 to 10 years, the usual test employed being that of Terman and Merrill. It was found that 59 of the children had an I.Q. of 50 or below, 24 an I.Q. of 50 to 75, 13 one of 76 to 94, and 5 an I.Q. of 100 or above. While only 4 children, including 3 with cerebral palsy, fared worse than expected, 16 fared better.

G. de M. Rudolph

(Editors's note—*This paper, which demonstrates the prognostic usefulness of Gesell testing in the first year of life, will be read with profit by all paediatricians and by all child psychologists.*—R. C. Mac Keith.)

The Formation and Extinction of Conditioned Reflexes in "Brain-damaged" and Mongoloid Children

H. G. BIRCH and H. DEMB. *Journal of Nervous and Mental Disease* Aug. 1959. 129, 162-170.

At the Morris Solomon Clinic for Retarded Children (The Jewish Hospital), Brooklyn, New York, the authors have studied 18 children considered to be "brain damaged", a term they apply to patients with a behaviour syndrome and clinical signs of trauma to the central nervous system but no anatomical lesion, 10 of these being classified as hyperactive and/or distractible and the others as non-hyperactive, and have compared the findings with those in 8 mongoloid and 4 normal children in relation to the production and extinction of a conditioned reflex, consisting of a light followed by a mild electric shock.

It was found that only 6 of the 10 hyperactive children could be conditioned and they scored on the same level as the mongols, while non-hyperactive patients performed

like the normal children. The level of performance was found to be unrelated to the I.Q. An interesting observation was that the hyperactive group were restless and easily distracted at all sittings, and even the 6 subjects who were conditioned did not lose the conditioned reflex by the normal method of internal inhibition, but by increasing motor activity till external inhibition resulted. Half the normal and mongoloid children fell asleep during the extinction process.

The authors suggest that the hyperactive group would probably function best in quiet surroundings and with stimuli kept at a low level and repetitive in type, while the others require constant stimuli to prevent generalized inhibition.

Janet Q. Ballantine

The External Cranial Volume of Normal Children. (In English)

J. B. JORGENSEN, E. PARIDON and E. QUADE.
Acta paediatrica July 1959. **48**, 371-378.

Simple measurement of the circumference of the head is a crude and inaccurate method of assessing the volume of the brain or of the cranial capacity in children. Working at the University of Copenhagen, the authors have therefore devised a method in which the child's head is immersed in water and the volume of the water thus displaced is measured. This is termed the external cranial volume (E.C.V.). The child is held supine and the head immersed to a line joining the glabella and the external occipital protuberance. Altogether 215 normal children aged 0 to 7 years have been examined and normal values obtained, with standard deviations, in relation to age, weight, total height, and standing height up to the tip of the ear. It is believed that this method of

assessment will help in the more accurate and earlier diagnosis of hydrocephalus and microcephaly.

(It would have been helpful if the results had been tabulated instead of being expressed graphically.)

John Lorber

The External Cranial Volume of Macro- and Microcephalic Children. (In English).

J. B. JORGENSEN, E. PARIDON and E. QUADE.
Acta paediatrica Sept. 1959. **48**, 469-476.

The authors report the results of measurement of the E.C.V. of 16 children in whom micro- or macro-cephaly was proved or suspected. In 2 children with undoubted hydrocephalus the values were far outside the normal range; in 4 children in whom lesser degrees of hydrocephalus were suspected, but unproven, the values were at the upper limit of normal or just above it. Of 3 cases of presumed microcephaly, the values were within the normal range in 2 and well above normal in one, whereas in 2 obvious microcephalics they were more than twice the standard deviation below the normal range. In 5 mongols the E.C.V. was within the normal range. It is concluded that this method of measurement is unnecessary in obvious cases of hydrocephalus or microcephaly, but may be of value in the early diagnosis of suspected cases.

(As the number of children who were investigated was very small and the diagnosis of the presumed lesser abnormalities was not correlated with ventriculography, these conclusions should not be accepted without further and more extensive investigations on a larger series of cases.)

John Lorber

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